

Sir Thomas Lewis

A PRIMER OF CARDIOLOGY

BY

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Second Edition, Thoroughly Revised, with 215 Illustrations



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DEDICATED

TO

VIVIAN GERARD BURCH

with Gratitude and Appreciation for Her Inestimable
Assistance in All of My Endeavors

PREFACE TO SECOND EDITION

As indicated in the previous edition this primer is intended for the medical student and physician who are interested in an introduction to cardiology. It is not encyclopedic but treats selected aspects of cardiology to illustrate the importance of applying fundamental principles of physiology to clinical cardiology in order to understand diseases of the heart. The

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New Orleans

G. F. B.

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A PRIMER OF CARDIOLOGY

Chapter 1

GENERAL ANATOMIC CONSIDERATIONS

A KNOWLEDGE of certain anatomic facts is essential in cardiology. Although it is obviously desirable that this knowledge be as detailed as possible, only some of the more general and essential anatomic principles can be presented in this limited space. This will be done primarily through the medium of diagrammatic illustrations.



FIG. 1. The angles formed by the longitudinal axis of the heart with the horizontal and midsagittal planes viewed in the frontal plane of the body.

CARDIAC AND THORACIC TOPOGRAPHY

It is extremely important that the relationship of the heart and its various components to the thorax be kept clearly in mind. This is necessary not only for the proper appreciation of the problems of physical diagnosis but especially for roentgenographic and electrocardiographic studies. Surgical and medical considerations require an adequate knowledge of topographic cardiac anatomy. It is not difficult to visualize clearly the position of the heart in the thorax. It should be remembered that it is suspended by the

APPENDIX

various anatomic components and t

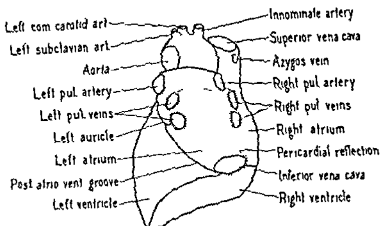


Fig. 4.—The relative position of the essential structures of the heart viewed posteriorly. The right ventricle is *anterior* and *inferior* (diaphragmatic).

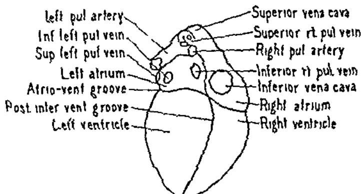


Fig. Diaphragmatic and posterior view of the heart. The right ventricle forms the greater part of the diaphragmatic surface of the heart.

These figures should be studied carefully and learned thoroughly. For example it is commonly erroneously *considered that* the right border of the heart is formed by the right *ventricle or that* the right ventricle is to the right. A glance at figure 3 shows *that the right* atrium forms the right

great vessels at the base in such a manner that the apex or longitudinal axis is directed *downward*, *forward* and *to the left* (Figs. 1 and 2).

The three important angles formed by the longitudinal axis of the average human heart with the *frontal*, *midsagittal* and *horizontal* planes of the body should be known.

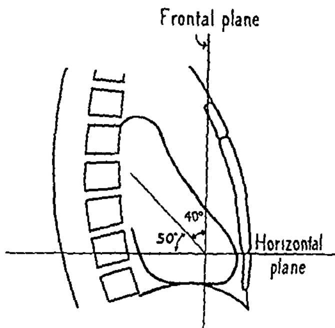


FIG 2.—The angles formed by the longitudinal axis of the heart with the horizontal and frontal planes viewed laterally

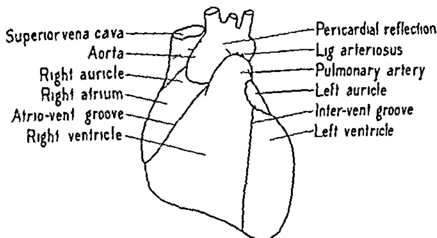


FIG. 3.—Diagrammatic representation of the main structures of the heart viewed anteriorly. Note the relative positions of the cardiac chambers For example, the right border of the heart shadow is *not* formed by the right ventricle, but rather by the right atrium.

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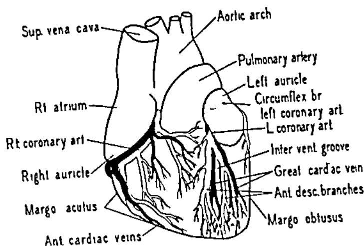


Fig 7 — Cardiac circulation noted anteriorly

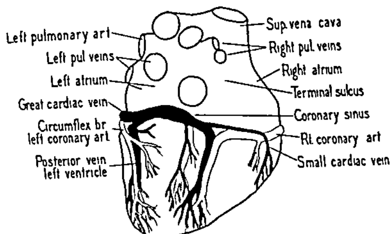


Fig 8 — Cardiac circulation noted posteriorly

. . . of the . terior *surface* of the heart are indi- . . . presenting themselves for con- . . . ocardial infarction trauma to . . . The larger coronary . . . are likewise of clinical . . . entricular (AV) node,

border of the heart and that the *right ventricle is situated anterodiaphragmatically* These and many other anatomic facts should be correctly learned before cardiology is approached

THE GREAT VESSELS AT THE BASE OF THE HEART

The large arteries and veins leaving and returning to the base of the heart are extremely important in cardiology The anatomic relationships of these structures (Fig 6) to each other and to other structures in the superior portion of the mediastinum should be permanently impressed upon one s mind For example inspection of figure 6 reveals how readily an aneurysm of the aortic arch may press upon the left recurrent laryngeal nerve and produce hoarseness and how an aneurysm of the superior portion

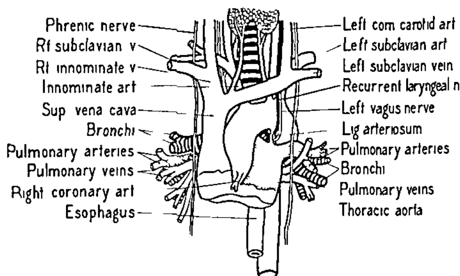


FIG 6 —The relative positions of the great vessels at the base of the heart Note *the left recurrent laryngeal nerve looping under the arch of the aorta* Because of this ... The close approximation and inter... to disturbances in ... n

... the left *thorough* *medias* *which* ... *otherwise appear complicated to those unfamiliar with such anatomic facts*

THE CARDIAC CIRCULATION

A knowledge of the present day concept of the circulation to the heart is not difficult to attain Many of the details of the circulation remain

. . . . . . will assist in a better . . . . surgery.

congenital anomalies and heart block . .

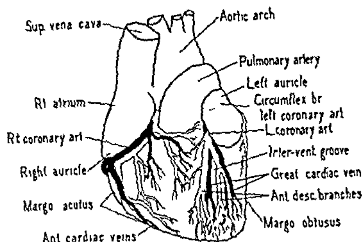


FIG 7 —Cardiac circulation noted anteriorly

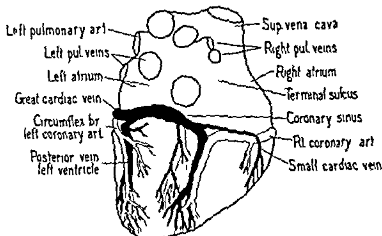


FIG 8 —*Cardiac circulation noted posteriorly*

The main coronary vessels of the *anterior surface* of the heart are indicated in figure 7 These vessels are forever presenting themselves for consideration in problems concerned with myocardial infarction, trauma to the heart, surgical therapeutic procedures and the like The larger coronary vessels of the *posterior surface* of the heart (Fig 8) are likewise of clinical importance It is well to remember that the atrioventricular (AV) node,

bundle of His and origin of the two bundle branches receive most of their arterial circulation from the posterior or right coronary artery. The anterior and posterior coronary arteries anastomose freely in the interventricular septum.

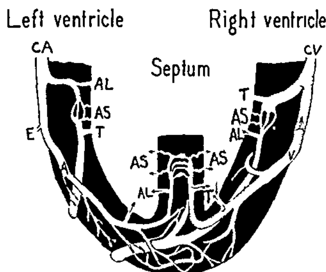


FIG. 9.—The circulation to the subendocardial myocardium by way of the lumina of the ventricles. These channels are concerned significantly with the development of collateral circulation. *CA*, coronary artery; *CV*, coronary vein; *T*, thebesian vein; *AL*, arterioluminal; *AS*, arteriosinusoidal; *E*, extracardiac vessel.

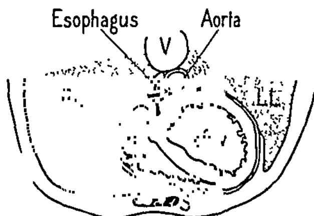


FIG. 10.—Transverse section of the heart and thorax showing the relative positions of the principal mediastinal structures and the ventricles.

The ventricular myocardium depends for nutrition, to a considerable degree, upon circulation by way of the lumina of the ventricles. This is particularly important under stresses of disease states concerned with impaired coronary artery circulation. The quantitative and qualitative

roles of the arterioluminal, thebesian and arteriosinusoidal vessels (Fig. 9) remain essentially unknown. Because of these vessels the subendocardial layers of ventricular muscle rarely succumb to coronary occlusion which often results in massive death of subepicardial muscle. These vessels seem to be concerned significantly with angina pectoris, a clinical state little known anatomically or physiologically.

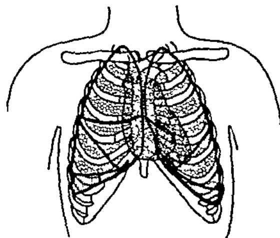


Fig. 11. The relationship of the heart to the pleural fissures and lobes of the lungs viewed anteriorly. The indicated pleural reflections connected by the parallel lines are the respective positions during inspiration and expiration.

CERTAIN CLINICAL ASPECTS OF CARDIAC ANATOMY

There are certain other relationships of the cardiac structures to the thoracic wall which have clinical significance. The relative positions of

The relationship of the heart to the lobes of the lungs and the fissures and pleura (Fig. 11) is extremely important. This is particularly well exemplified by the clinical problems concerned with pericarditis. Not infrequently pneumonitis or pleuritis near the heart results in septic or sterile pericarditis. These
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might evade the pulmonary parenchyma and pleural spaces as well as

[illegible] The anatomic valvular areas may be covered by a silver dollar

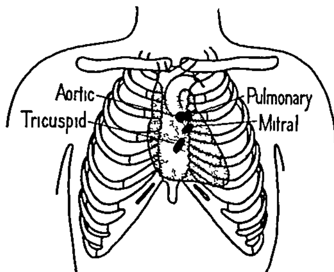


Fig 12 — The *anatomic* valvular areas of the heart noted topographically on the anterior surface of the thorax

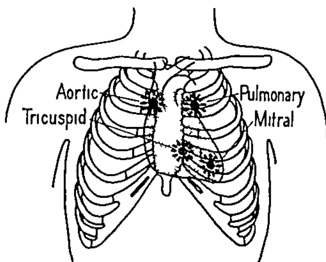
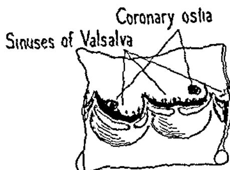


Fig 13 — The *clinical* valvular areas noted topographically on the anterior surface of the thorax It is at the points indicated that the auscultatory physiologic valvular phenomena are most intense Compare these areas with the *anatomic* ones shown in figure 12

whereas the clinical valvular areas are widely separated The striking differences in distribution of the clinical valvular areas are due to physical problems concerned with the conduction and production of the heart sounds This is explained partially by the fact that all sound usually considered as resulting from valvular closure actually is the result of other events, such as snapping of the chordæ tendineæ and blood movements

In addition the sound is not conducted directly outward but through conducting tissue or media of varying sound transmission characteristics including blood in the blood vessels and air in the lungs. In large part because of anatomic factors the characteristics of the heart sounds that reach the observer's ears are considerably different from those at the source. During auscultation of heart sounds and murmurs these facts should be kept in mind.



Fi 11 ... cut longitudinally with the interior surfaces of the ... of Valsalva ... exposed. One cusp has been severed in order to expose the interior of the ...

THE CARDIAC VALVES

The structure and detailed anatomic characteristics of the cardiac valves should be learned thoroughly but will not be discussed in detail here. Many clinical states are dependent upon anatomic peculiarities of these valves. Functional states of the valves which are of considerable importance and interest are dependent upon anatomic characteristics. Many structural configurations in the heart must possess definite useful evolutionary peculiarities which are not chance occurrences but are the result of well organized developmental processes. Such evolutionary developments resulting in anatomic states of importance in ensuring physiologic efficiency are well exemplified by the aortic valves (Figs. 14, 15 and 16). Two interesting examples are described and of course it is possible to analyze many others.

1 The essentially saccular configuration of the ... Valsalva from which ... the efficiency

of t ... volume of blood flow. This phenomenon is particularly likely to occur with sudden clo... valves. In the presen...

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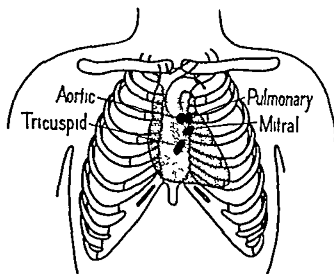


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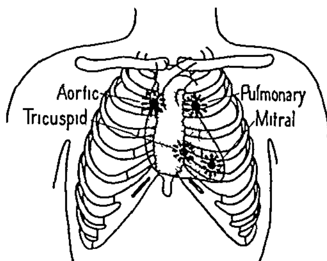
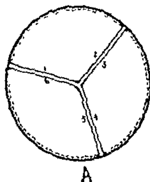
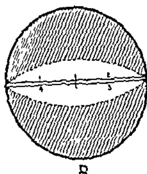
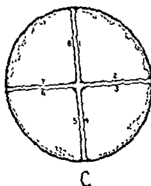


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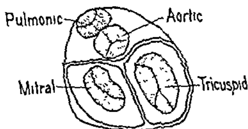


FIG 17 —Supraventricular view of the closed valves of the heart All valves are closed simultaneously, and for a moment physiologically, during the period of isometric relaxation of the cardiac cycle

wider opening of the ostia of the coronary arteries. These phenomena illustrate the *dynamic nature* of cardiac anatomy.

2. The presence of *three cusps* to guard the aortic and pulmonic valves must not be merely a chance phenomenon, for *any number* of cusps other than three would result in less efficiency of blood flow through the two openings (Fig. 16). It can be seen from figure 16 that with the nature of the construction of the aortic and pulmonic valves their free edges total a length which is essentially equal to six times the radius (6 r) of the aortic or pulmonic orifices. This is also essentially equal to the circumference of the aortic or pulmonic rings or openings. Therefore, when these valves open

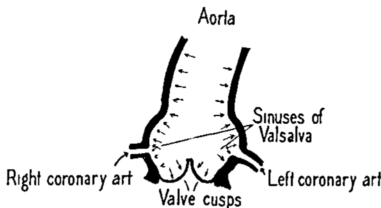


Fig. 15. The saccular shape of the sinuses of Valsalva tends to ensure more efficient coronary blood flow. With an increase in pressure in these sinuses there is distention of the walls with a tendency for the walls to pull away from the ostia of the coronary arteries. This tends to result in an increase in size of these ostia and in turn causes more blood to flow to the myocardium. This is particularly likely to occur with sudden closure of the aortic valves, especially in diastolic hypertension.

the cusps rest smoothly and snugly against the wall of the roots of the aorta and pulmonary artery, ensuring maximal opening of the orifices. If there were two cusps the length of the edges of the valves would total 4 r or essentially two thirds the length of the circumference of the aortic and pulmonic orifices respectively. Stenosis of the openings would result. If there were more than three cusps, for example four, the total length of the edges of the cusps would exceed the circumference of the orifices, and therefore folding and overlapping of the cusps would occur when the valves open. This would also result in stenosis of the openings. This further illustrates the peculiarities of cardiac anatomy.

Chordae tendineae and papillary muscles are peculiar to the mitral or left atrioventricular (two leaflets) valve and the tricuspid or right atrioventricular (three leaflets) valve. This fact is particularly important for a better understanding of functional insufficiency of these valves. Figure 17 shows the supraventricular appearance of the four heart valves when they are all closed simultaneously, a phenomenon occurring during the period of isometric relaxation.

It should be noted that most of the early impulses to the septum are transmitted by the left bundle branch

an

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and certain irregularities It should be thoroughly learned

THE CONDUCTION TISSUE

The conduction tissue is a highly specialized type of muscle which transmits impulses with relatively great rapidity. The sinoauricular (SA) node is the normal pacemaker of the heart, that is, it is the site of impulse formation. The atrioventricular (AV) node ensures proper asynchronous contraction of the atria and ventricles by delaying the cardiac impulse from the atria for an adequate length of time to ensure proper timing of ventricular contraction, thus resulting in efficient cardiac hemodynamics. The bundle of His, bundle branches and Purkinje system provide adequate and prompt distribution of the impulse to the ventricular musculature (Fig 18).

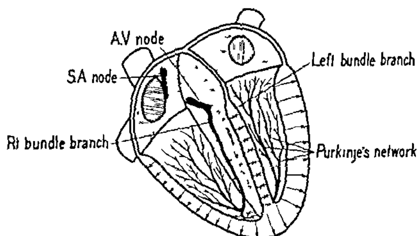


Fig 18 — Diagrammatic representation of the conduction tissue of the heart

In brief, the conduction tissue is distributed as follows. The sinoauricular node is located on the posterior wall of the right atrium near the entrance of the venæ cava. Conduction from this node occurs across the atrial musculature to the atrioventricular node located posteriorly near the junction of atrial and ventricular septa. Of course transmission of the impulse occurs throughout the atrial musculature but is prevented from continuing into the muscle of ventricles by a nonresponsive fibrous ring. This ring constitutes the skeletal framework of the heart and supports the valves and great vessels. The only normal pathway into the ventricles is through the bundle of His, which extends into the interventricular septum, at which point it divides and continues down each side of the interventricular septum posteriorly to the respective inner ventricular surfaces. Its structure is actually similar to a bundle, and as it leaves the septum it often courses as a discrete band across the cavity of the heart, especially on the right (*moderator band*). As it spreads out along the ventricle, it branches and finally divides into the terminal arborizations (Purkinje system). The mode of radiation is shown by the small arrows (Fig 18).

rheumatic fever, anemia, hypertension, arteriosclerosis, syphilis and the like cannot be detected

2 **Heart Disease** is said to be present only when a *reliable or pathognomonic sign* of heart disease has been elicited It is extremely important to be al [illegible]
served l [illegible]
of heart [illegible]
portant cardiac neuroses are [illegible]
disease merely as a result of [illegible]
diagnosis of heart disease is [illegible]
psychic disturbances with some degree of unhappiness and anxiety Furthermore a patient who has once been *given an erroneous diagnosis of* heart disease can never be completely convinced that his heart is normal, even when rendered such an opinion by experts in the field *Therefore one must be sure of the diagnosis before telling a person he has heart disease*

3 **A Diagnosis of Possible Heart Disease** is made in a patient *who has signs and symptoms which suggest heart disease, but in whom no definite criterion of heart disease can be elicited* At the time the patient may or may not have an etiologic factor known to produce heart disease For example, a patient with hypertension of recent development vague precordial [illegible]

[illegible] **Potential Heart Disease** is said to be present in a patient *with an etiologic factor known to produce heart disease but in whom there are no signs symptoms or other clinical data indicative or suggestive of heart disease* [illegible]
examp [illegible]
sugg [illegible]
thme [illegible]
Dise [illegible]

THE DEPENDABLE OR PATHOGNOMONIC SIGNS OF ORGANIC HEART DISEASE

As stated previously before a diagnosis of organic heart disease can be made it is necessary that at least one definite criterion for heart disease be found Any one or more of the signs listed may be considered as reliable signs of heart disease All of these are relatively easy to detect under the usual clinical circumstances or entirely at the bedside The special circumstances in which each sign may possess less reliability will be discussed

1 Cardiac enlargement
2 Auricular fibrillation
3 Auricular flutter
4 Heart block
5 Pulsus alternans
6 Pericardial friction rub

Chapter 2

APPROACH TO THE DIAGNOSIS OF HEART DISEASE

In order to make an intelligent and complete diagnosis of heart disease it is essential that the problem be approached in an organized fashion. In realization of this fact the New York Heart Association and American Heart Association have published an excellent little book *Nomenclature and Criteria for the Diagnosis of Diseases of the Heart*. All students and physicians who are concerned with the heart to the slightest extent should not only own but should study, learn and understand its contents. It will be assumed in the following discussions that the contents of this book have been thoroughly mastered. A proper approach to the study and diagnosis of the patient will yield a more complete inventory of his cardiac state, and consequently his management will be considerably more satisfactory. A superficial study will result in superficial management; a detailed thorough study will lead to satisfactory management with proper consideration and evaluation of all details. Careful organized procurement of the cardiac inventory also permits anticipation of complications and more accurate prognosis.

FOUR MAJOR CLINICAL DIAGNOSTIC CARDIAC CATEGORIES

Whenever a person presents himself for an examination and a cardiac evaluation is to be rendered, all possible available methods of study should be utilized in the clinical workup. Experience has shown that even after all available methods have been adequately employed the evaluation is not absolutely satisfactory; therefore no method of study, however minor, should be omitted. The heart of every person, once it has been completely studied, can be classified in one of four *major clinical diagnostic cardiac categories*:

1. No heart disease,
2. Heart disease,
3. Possible heart disease, or
4. Potential heart disease.

The *criteria* necessary for the diagnosis of each of these must be known and fulfilled in order to render the *correct* diagnosis. The *diagnostic criteria* for each of the foregoing categories are as follows:

1. **No Heart Disease.** A person is said to have no heart disease or normal heart *if there are no symptoms or signs which are indicative or suggestive of heart disease and if at the same time there is no active etiologic factor present known to cause heart disease*, i.e., active clinical states such as

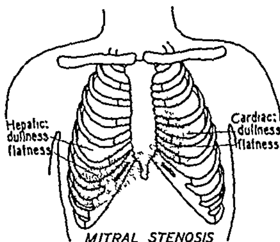
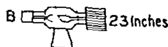


Fig 20 —The area of cardiac dulness and flatness is widened over the base of the heart in mitral stenosis The area of dulness is increased in area to the left and upward





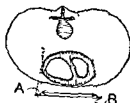


Fig 21 The proximity of the source of the [illegible]

7. Diastolic murmur
8. Thrill
9. Generalized senile arteriosclerosis
10. Thyrotoxicosis of several months' duration
11. Myxedema
12. True Heberden pain (angina pectoris and myocardial infarction syndromes)
13. Symmetric and definite elevation of venous pressure in all veins throughout the body
14. Diastolic hypertension for a prolonged period of time
15. Severe anemia
16. Protodiastolic gallop rhythm
17. Many definite electrocardiographic changes

1. **Cardiac Enlargement.** Any heart that is enlarged is abnormal. The possibility of "normal athletic enlargement" is still controversial, however. From the practical clinical point of view any localized or generalized cardiac enlargement indicates heart disease.

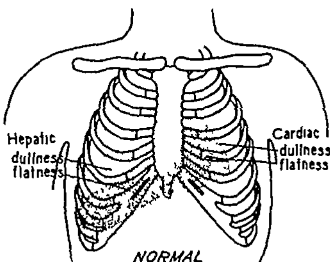


Fig. 19.—The area of cardiac and hepatic dullness (light stippling) and flatness (dark stippling) in the normal subject.

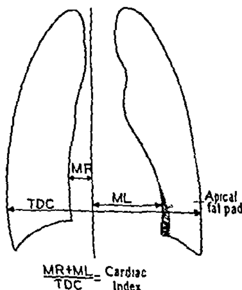
Cardiac enlargement is *detected* by:

(a) *Inspection of the precordial region.* With enlargement the apical pulsation or the point of maximal impulse (PMI) is displaced to the left and downward beyond the midclavicular line and usually below the level of the fifth intercostal space. With extreme enlargement the precordial region may bulge outward; this is particularly true in children with right ventricular enlargement.

(b) *Palpation of the cardiac region* will reveal the position of the PMI and apex beat. With left ventricular enlargement the PMI is displaced downward and to the left. Bulging of the anterior chest wall can also be palpated.

Teleoroentgenography is the most frequently employed of the roentgenographic methods of examination. It has advantages of being simple and fairly inexpensive. Furthermore, it results in a permanent record which is available in the future for comparative purposes to follow the course of variations in cardiac size and configuration.

In addition to the PA (erect postero-anterior) view (Fig. 24), views are taken in the left anterior oblique, right anterior oblique and lateral positions (Figs. 25-26). The right anterior oblique view permits examination of the root of the aorta, pulmonary conus, main trunk of the pulmonary artery, right ventricle, left atrium and esophagus. The left anterior oblique view (Fig. 26) permits study of the entire thoracic aorta from the root to the diaphragm, esophagus, posterior surface of the heart, pulmonary arteries, right ventricle and inflow tract of the left ventricle. A barium sulfate (radiopaque) paste should be swallowed by the patient to permit visualization of the configuration and position of the esophagus, and teleo-



Fi[illegible] D[illegible]

is made the ap[illegible]

MR = [illegible] clinical error

MI [illegible]

MR + [illegible]

TDC

The m[illegible]

(c) *Percussion* enables the cardiac area of dulness to be outlined By comparison of the area percussed with the expected normal size the degree of enlargement can be evaluated (Fig 19) In mitral stenosis there may be enlargement to the left and upward without displacement of the apex (Fig 20)

(d) *Roentgenography* Unfortunately, the methods employed in physical examination are unreliable and often grossly inaccurate, especially with borderline cardiac size and with pulmonary disease and thoracic deformities Roentgenographic methods are the methods of choice for the study of cardiac size and configuration There are several types of examination available, the most useful of which are

(1) Teleoroentgenography
(2) Fluoroscopy
(3) Orthodiagraphy
(4) Roentgenokymography
(5) Angiocardiography
(6) Planography

(1) [illegible]
place [illegible]
vent [illegible]
with the anterior surface of his chest against the cassette and with the x-ray tube placed posteriorly If the source of the roentgen rays is placed near the heart, magnification of the cardiac outline results The closer the heart is to the source of the x-rays, the greater the magnification (Fig 21)

Many types of measurements and standards have been developed Most of them are highly detailed technical and impractical, and add little information to the more practical ones employed generally Under most clinical conditions, the simple ratio of the transverse diameter of the heart to the greatest internal diameter of the chest proves to be most practical These measurements are obtained directly from the teleoroentgenogram (Fig 22 and Appendix) The transverse diameter of the heart is obtained by drawing a midsternal line through the center of the sternum on the teleoroentgenogram A line perpendicular to this line is extended to the furthermost right margin of the cardiac shadow Another line perpendicular to the midsternal line is extended to the outermost margin of the cardiac shadow to the left Care must be taken not to include the apical fat pad (Fig 22) The sum of the lengths of these two lines represents the *transverse diameter of the heart* *The greatest internal diameter of the chest* is measured in the same units of length In the normal heart the transverse diameter of the heart should not exceed 57 per cent of the greatest internal diameter of the chest Clinicians usually employ the value 50 per cent instead of 57 per cent because of greater simplicity Any value greater than 57 per cent indicates definite enlargement, a pathognomonic sign of heart disease Unfortunately, this method of measurement is subject to error, *i e*, a heart may enlarge a great deal before the cardiac index reaches abnormal values This is particularly true in patients with ptotic ("dropped") hearts (Fig 23) The Ungerleider Tables (see Appendix) are considered by some as a more accurate index of cardiac size

Teleoroentgenography is the most frequently employed of the roentgenographic methods of examination. It has advantages of being simple and fairly inexpensive. Furthermore, it results in a permanent record which is available in the future for comparative purposes to follow the course of variations in cardiac size and configuration.

In addition to the EPA (erect posteroanterior) view (Fig. 21) views are taken in the left anterior oblique, right anterior oblique and lateral [illegible]. The right anterior oblique view permits examina- [illegible]

diaphragm, esophagus, posterior surface [illegible] arteries, right ventricle and inflow tract of the left ventricle. A barium sulfate (radiopaque) paste should be swallowed by the patient to permit visualization of the configuration and position of the esophagus, and teleo-

$$\frac{MR+ML}{TDC} = \text{Cardiac Index}$$

Fig. 22. D[illegible] [illegible] th[illegible] [illegible] the [illegible] posterior [illegible] heart and with the [illegible] the greatest transverse [illegible] the greatest transverse internal [illegible] chest. When a measurement of the transverse diameter of the heart [illegible] the apical fat pad should not be included. This is a common technical error.

MR = maximal distance from midline [illegible]

ML [illegible]

MR + [illegible]

TDC [illegible] internal diameter of the chest.

The unit of measurement is usually centimeters (see Appendix).

roentgenograms should be taken in three or more views. This permits further analysis of changes in cardiac size and distortions (Fig. 27).

(2) *Fluoroscopy.*—Fluoroscopic examination is the most informative of all the roentgenologic methods. The patient is placed between the fluoroscopic screen and the x-ray tube and is examined in a dark room. The nature of the movements of the chambers of the heart and great vessels is studied. To examine the heart properly, the examiner should remain in an absolutely dark room for at least twenty minutes before beginning

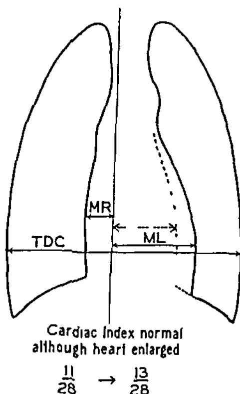


Fig. 23. Diagrammatic representation of a *ptotic heart* with a $\frac{MR + ML}{TDC} = \frac{11}{28}$ at one time. Later the heart increased in size from the dotted line to the solid line and $\frac{MR + ML}{TDC}$ increased to $\frac{13}{28}$. Although the heart increased in size, the cardiac index remained within normal limits. This type of observation indicates (1) the need for serial teleoroentgenograms and (2) the shortcomings of the cardiac index in the determination of cardiac enlargement.

the fluoroscopic examination. *One of the greatest errors which leads to* poor fluoroscopic examination is failure to allow for full accommodation for vision in a poorly illuminated room. During fluoroscopic examination the beating heart is observed while its position is varied by rotating the patient. The patient should then swallow a *thick paste* of barium sulfate

a radiopaque material This thick paste adheres to the mucosa of the esophagus and permits adequate visualization of the esophagus including distortion and displacement of various portions by the extra-esophageal structures such as the heart and great vessels This should be done with the patient first in the right anterior oblique position and then in other positions Any displacement that results from disturbances in cardiac size

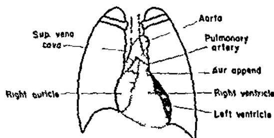


Fig 24 —The cardiac silhouette as observed in the *anteroposterior position* These anatomic relationships are important in fluoroscopic and teleroentgenographic studies This view is particularly useful in a study of the outflow tract of the left ventricle the pulmonary conus and the aortic knob

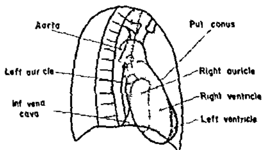


Fig 25 —The
This position is p
right ventricle pu

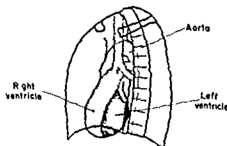


Fig 26 —The cardiac silhouette in the *left anterior oblique position* This is particularly useful for a study of the inflow tract of the left ventricle the aortic arch and descending aorta, the pulmonary tissue behind the heart and the pulmonary artery

can be detected (Fig 27) Unfortunately the cardiac size cannot be permanently recorded fluoroscopically, although it can be described and recorded fairly accurately by this method Furthermore, because of the relative positions of the x ray tube patient's heart and fluoroscopic screen the heart size is variably magnified (Fig 21) With experience the observer can acquire skill in evaluating the degree of magnification due to the fluoroscope so that proper allowances can be made By means of the fluoroscope variations in the degree of pulsations paradoxical pulsations in cardiac aneurysms tugging by adhesions reduced pulsations in pericardial effusion etc , can be observed and evaluated

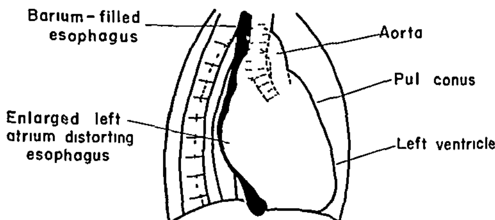


Fig 27 —The cardiac silhouette in the *right anterior oblique position* showing the dilated left atrium displacing the barium filled esophagus in *mitral stenosis*

(3) *Orthodiagraphy* is employed relatively infrequently in this country It permits not only a fluoroscopic study but also an accurate and permanent record of cardiac size and configuration without magnification In this method the fluorescent screen remains stationary while the x ray tube is moved by the examiner *in order to bring the central beam of x rays emitted* perpendicular to the fluoroscopic screen and in direct line with the borders of the heart thus only a central stream of x rays moving in a straight non diverging line is used (Fig 28) A tracing of the cardiac silhouette is made on transparent paper placed on the fluorescent screen Tracings may be made of any view desired with or without the use of barium in the esophagus

By means of a suitable lever and locking device the orthodiagraphic mechanism with the fixed screen and independently movable x ray tube can be converted to a fluoroscopic mechanism in which the screen and tube move simultaneously as a single unit

(4) *Roentgenokymography* is not employed frequently although it has valuable applications in clinical cardiology For recording the roentgenokymogram the same procedure is employed as for a teleroentgenogram except that the cassette containing *the film is moved steadily and relatively slowly* for about 11 millimeters in front of a series of horizontal lead lattices

placed 12 millimeters apart The resultant record (Fig 29) shows a serrated border with the crests being the edge of the heart shadow at the termination of diastole and the troughs the border at the end of systole If it were not for the fact that the heart shifted and rotated in position the relative distances of peaks and troughs would be an accurate index of the stroke volume The roentgenokymogram is of some use, nevertheless in *estimating* stroke volume It is especially useful in localizing an *aneurysm* or area of *infarction* of the heart In the infarcted area there is an

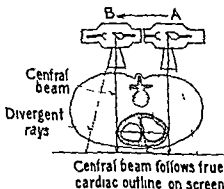


Fig 28 Diagram illustrating the manner in which only the central portion of the x ray beam is directed perpendicular to the fluoroscopic screen in outlining the cardiac silhouette The x ray tube is moved while the fluoroscopic screen is fixed The x ray tube is moved along the outermost margin of the heart thus casting a true shadow of the heart on the fluoroscopic screen for tracing This type of roentgenologic study is known as *orthodiagraphy*

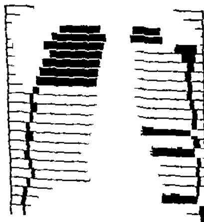


Fig 29 —Diagram of a *roentgenokymogram* Consult text for details

outward bulging during systole, or a *crest* instead of a *trough* as the dead muscle fails to contract and the high intraventricular pressure occurring during systole forces the infarcted zone outward (Fig. 30). It is also of value in demonstrating pericardial fluid which reduces the amplitude of the peaks *and troughs and for identifying various chambers of the heart and great* vessels by the relative timing of their phases of expansion and contraction

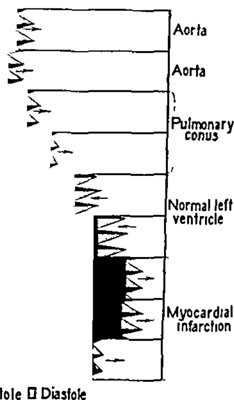


Fig. 30—The left lateral portion of a roentgenokymogram in a patient with an anterolateral infarct to show the details of the discordant pulsations in the various portions of *the heart and great vessels. On close inspection of the volume pulsations* ... left ventricle contracts ... is true for the pulmon[illegible]

ventricles

(5) *Angiocardiography*—The vessels near the heart and the chambers of the heart may be observed roentgenographically for variations in size or for congenital and acquired anomalies by means of radiopaque materials injected into the blood stream *followed immediately by serial roentgeno-* [illegible]

into a vein or may be instilled by means of a cardiac catheter into any chamber or great vessel of the heart. Instillations on the arterial side may be associated with serious reactions. This form of visualization of the heart and its great vessels has rather limited applications at present. It is employed only for special problems. Whenever a safe radiopaque material is developed, it will probably be used more generally.

(6) *Planography*.—This type of roentgenographic study involves, by special means, visualization of selected planar depths of the heart upon x-ray film. It is employed particularly in cardiology for the search of calcified areas of the heart, such as calcification of valves, areas of the pericardium or zones of myocardial infarction.

2. **Auricular Fibrillation**.—Auricular fibrillation should be considered a *definite sign of heart disease until proved otherwise*. True enough, young

... an individual into a cardiac neurotic. When auricular fibrillation is the *principal* abnormal manifestation, the physician should never suggest to the patient that he has heart disease until he is certain of the diagnosis. Many of the cardiac states associated with auricular fibrillation are reversible; therefore, a diagnosis of heart disease should be withheld from the patient until proved to exist. In the *presence of a cause for heart disease* and in older people, auricular fibrillation is a good sign of heart disease.

3. **Auricular Flutter**.—The same rules hold as indicated for auricular fibrillation. Consult the section on cardiac irregularities for further considerations of these two types of ...

4. **Heart Block**.—*Heart* ...
sign of heart disease. I...
clinical diagnosis is indefi...
recording device. Heart block is discussed in mor...

... it is necessary ...

... cannot be stopped. Pulsus alternans may follow *digitalis* intoxication and disappear when the drug is reduced to proper doses. Such an altern...

... heart disease. It is well to remember that an alternation of only 2 or 3 mm. mercury may exist and be missed if the blood pressure is not determined properly. It seems advisable to discuss here certain aspects of the *method for recording the arterial blood pressure* since there is a great tendency to determine the arterial blood pressure improperly.

outward bulging during systole or a *crest* instead of a *trough* as the dead muscle fails to contract and the high intraventricular pressure occurring
... also of value
the peaks
and great
vessels by the relative timing of their phases of expansion and contraction

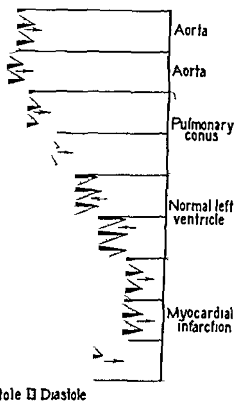


Fig 30 —The left lateral portion of a roentgenokymogram in a patient with an anterolateral infarct to show the details of the discordant pulsations in the various portions of the heart and great vessels On close inspection of the volume pulsations
left ventricle contracts
is true for the pulmon

ventricles

(5) *Angiocardiography*—The vessels near the heart and the chambers of the heart may ... variations in size or for congenital a ... diopaque materials injected into the ... serial roentgeno graphic films or ... The radiopaque material, usually a concentrated organic solution of iodine is injected rapidly

greater clinical value Until such time both diastolic blood pressures should be recorded Such a blood pressure would be recorded as follows

$$\frac{160}{90-80} \quad \text{or } 160/90/80$$

160 = systolic blood pressure
90 = diastolic blood pressure at beginning of fourth phase
80 = diastolic blood pressure at beginning of fifth phase

In persons in whom the sounds do not disappear it will be found that the beginning of a fifth phase can be estimated, that is at a point when the fourth phase sounds show a definite change in intensity

It is extremely important to record and interpret blood pressure measurements properly Remember that arterial blood pressure is exceedingly and *necessarily* variable A recording at any one moment merely indicates the arterial blood pressure for that moment that is, when the patient is sitting near the physician with his arm on the doctor's desk This is no

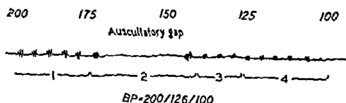


FIG 32 Diagram illustrating the *auscultatory gap* This is often a cause for error in careless recording of arterial blood pressure Consult the text

index of what it is when the patient is emotionally upset digging a ditch, running a dash or sleeping soundly in bed at 3 o clock in the morning It is unfortunate that a continuous recordin...

...ent when compared to a blood pressure that remains elevated night and day Furthermore a blood pressure of 160 80 in a patient with senile arteriosclerosis may be considered insignificant when recorded in th...
man runs for a street car it may
easily rupture a weakened vessel
the pressure at that moment o... physician wants
variations of the
he may be adeq...
likely to produce
vascular accidents ... can result in fatal cardio-
For

Measurement of the Arterial Blood Pressure.—The patient should be seated comfortably and relaxed. There should be no constricting clothing about the arm or limb to be examined. The standard blood pressure cuff (12 cm. in width) is wrapped snugly around the slightly abducted arm. The pressure within the cuff is rapidly elevated to 250 to 300 mm. of mercury and the radial artery is palpated (*palpatory method*). The pressure at which the pulse at the wrist or ankle just returns is recorded as the systolic blood pressure. This procedure should always be employed first for orientation and, as a check of the next method (*auscultatory*). The pressure within the cuff is again raised quickly to 250 or 300 mm. of mercury and the region of the brachial artery at the antecubital space below the cuff is auscultated with a stethoscope, while the physician listens for the *Korotkov* sounds. The pressure within the cuff is slowly reduced until the

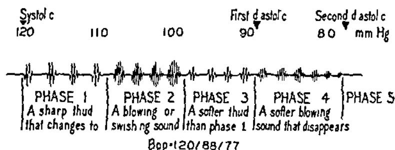


FIG. 31.—Diagram illustrating the *Korotkov sounds* and various phases observed during measurement of intra-arterial blood pressure. Consult the text for details.

first sounds appear (the beginning of *first phase*). The pressure (mm. mercury) in the sphygmomanometer at that moment is recorded as the *systolic blood pressure*. As the pressure in the pneumatic cuff is gradually reduced, the sounds then change (Fig. 31) from a sharp thud to a blowing or swishing sound (the *second phase*). With further slow reduction in pressure in the cuff the sounds then change again to a sharp sound or thud (*third phase*) that are not as intense as the sounds of phase one. The sounds then become blowing and relatively low in intensity (*fourth phase*). As the pressure in the cuff is reduced further, the sounds remain of the same general quality but become fainter and fainter and finally disappear (*fifth phase*). The final disappearance of the sounds may occur suddenly in some individuals or gradually or not at all in others. *The diastolic blood pressure* is recorded by some clinicians as the pressure within the cuff at the moment of change from the *third to the fourth phase* and by others, including our group, at the *change from the fourth to the fifth phase*, that is, when the sounds finally disappear. Which of these is more correct is not known. Clinical correlations and years of follow-up studies will finally indicate which has the

auricular node. Alternations in force of contraction due to bigeminy produced by premature contractions would not be considered true pulsus alternans. In true pulsus alternans it is noted that when the pressure in the blood pressure cuff is reduced to record the *systolic pressure*, the first phase with sharp sounds appears at a rate which is half the cardiac rate. As the pressure in the cuff is further reduced, the sounds double in rate or equal the cardiac rate in number. These later appearing sounds are of less intensity than the alternating ones that appeared at the higher pressure. All through the various phases, when the two groups of sounds exist together, they alternate in intensity. As the pressure in the cuff is further reduced and the pulsus alternans exists to a high degree, it will be found that the sounds of greater intensity pass through the various phases of the Korotkow sounds before the alternating ones of lesser intensity. Therefore, the sounds of the greater intensity pass into the *fifth phase* or *disappear* before those of lesser intensity (Fig. 33). The degree of pulsus alternans is expressed quantitatively as the difference in systolic pressure levels between the *appearance* of the two groups of sounds. For example, suppose the more intense sounds appear at a pressure of 180 mm of mercury and the weaker ones appear, or the sounds double in rate, when the cuff pressure is lowered to 160 mm of mercury, then the alternation is

$$180 - 160 = 20 \text{ mm mercury}$$

The patient is then said to have an alternation of 20 mm of mercury. The greater the pressure difference, the more severe the alternation and the worse the prognosis. *Normally* there is no alternation.

Pulsus alternans is frequently missed because it is too slight to detect by ordinary clinical practices when the pressure in the cuff is reduced too rapidly. Obviously, when an alternation of 2 or 3 mm of mercury exists, it is easily overlooked unless the cuff pressure is reduced slowly.

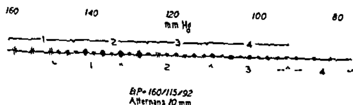


Fig. 33. Diagram illustrating the Korotkow sounds ... the alternate *more fo*... pressur... weaker

This requires effort, time and patience. Many measurements may have to be made before a stabilized level is attained.

During the recording of blood pressure certain *technical errors* may be encountered.

(*a*) The *auscultatory gap* (Fig. 32). In some subjects, for no apparent reason, the blowing sounds of the second phase may not be present for certain pressure levels within the cuff. This interval of no sound is known as the *auscultatory gap*. Should the pressure cuff be elevated insufficiently, that is, to within the level of the auscultatory gap, the appearance of *second phase* sounds at the lower pressure levels will be erroneously considered the onset of the first phase and recorded as the systolic blood pressure (Fig. 31). To avoid this error it is necessary: (1) always to inflate the blood pressure cuff to high levels and (2) to listen attentively to the character of the sounds that first appear as the cuff pressure is reduced. If they are not sharp, the determination should be *repeated* and the cuff should be inflated to a pressure of 240 mm. or more of mercury.

(*b*) *Relatively narrow cuff*. When the blood pressure cuff is too narrow, erroneously high pressures may be recorded. This is likely to occur in obese people with fat arms and legs. Special wide cuffs may be obtained for these people. If a wide cuff is not available, the regular one may be bandaged snugly in place (not to constrict the circulation) by first putting the cuff on and then wrapping it in place with ordinary 3 inch roller bandage.

(*c*) *Metal ribbed cuffs*. When the cuffs with metal ribs which snap in place are used, they must be placed properly. The arrows on the cuff *indicate where it should be placed in relation to the artery*.

(*d*) *Aneroid type of manometer*. The aneroid manometers frequently change in sensitivity and must be recalibrated. They should therefore be checked every six months against a mercury manometer.

(*e*) *Insufficient relaxation of the patient*. Patients are usually psychically disturbed when visiting their doctor, especially during a first visit. Pressures should therefore be recorded frequently at each visit and especially after quieting the patient. In order to interpret the significance of any one value, *the other determinations must be taken into consideration*.

(*f*) *Rapid reduction of pressure in cuff*. Remember that the cardiac cycle or the pulse wave lasts approximately 0.8 second at ordinary resting heart rates. *Therefore, if the pressure in the cuff is reduced rapidly, that is, several mm. of mercury between two beats, the true systolic or diastolic value will be missed*. Reduce the pressure slowly, make a tentative recording and repeat the procedure, reducing the pressure in the cuff especially slowly when the previously recorded values are approached. A true value will thus be obtained.

Now that some of the problems of recording the blood pressure have been discussed, *pulsus alternans* should be reconsidered. When a patient presents an alternating pulse, a cardiac systole of strong force alternates with one of weak or lesser force. The contractions *appear on time* as they must, for they are initiated by the same pacemaker, usually the sino-

and, dramatically, by nitroglycerin placed sublingually. It does not last over fifteen minutes and usually only three or four minutes.

The typical pain of *coronary occlusion* is of the same character and distribution but usually more severe and of much longer duration. The pain of myocardial infarction is not usually relieved by rest or nitroglycerine

often associated with

pulse and reduction

A friction rub, fever,

leukocytosis and an increase in the rate of sedimentation of the erythrocytes usually occur by the next twenty four hours and last a few days. The sedimentation rate is the last of these to return to normal.

When these syndromes are present in the classical picture there is no doubt that cardiac disease exists. However, about 30 to 35 per cent or more of the patients with these clinical states will not present a classical picture.

13. **Generalized and Symmetric Venous Hypertension.**—A definite symmetric and equal elevation of venous pressure in all veins is usually due to heart disease. The venous pressure should be elevated to at least 200 mm. water. This is associated with distended neck veins when the patient is erect. There is usually a large liver and there may be dependent edema, ascites or pleural effusion. These are most often due to *right ventricular congestive heart failure* but may be due to *concretio cordis*, *pericardial effusion*, *cardiac tamponade* from intrapericardial hemorrhage or a *network* of Chiari. Mediastinal tumors, adhesions, thrombosis of the superior and inferior venae cavae and the like are unlikely, if ever, to produce equal obstruction of both venae cavae and result in *equal* elevation in venous pressure in the tributaries of both.

Venous Pressure Measurements.—It is appropriate to discuss here certain methods of measuring venous pressure which have practical clinical application.

1. *Indirect Phlebomanometry.* (a) *Method of Lewis.* In the normal subject the external jugular veins are collapsed above the level of the supra

standing. If these veins are

In fact the distention of

under certain conditions indicates

increased venous pressure.

(b) *Method of Gaertner.* A *simple method* of measuring the venous pressure in the veins of the limbs is to lower the extended hand or foot slowly and allow the veins to fill, then raise them slowly until the veins just collapse. The level at which this collapse occurs can be located sharply if the veins are conspicuous. The difference between this level and the heart level or phlebostatic axis in millimeters is essentially the venous pressure in millimeters of blood in that vein. This method is simple and practical.

(c) *Method of* [illegible]

positi

of his

near his body. Nor-

An alternation of 2, 3 or even 5 mm. of mercury exists in *many* patients with left ventricular congestive heart failure, yet rarely does one encounter a clinical record with pulsus alternans noted. *Therefore* record all blood pressures *slowly, carefully, patiently*, and with a *consideration* equal to its importance. *Remember that pulsus alternans is of extreme clinical importance in diagnosis, prognosis and therapy.*

6. **Pericardial Friction Rub.**—A pericardial friction rub is a definite sign of heart disease, as it indicates a diseased pericardium, which is an extremely important part of the heart. Care must be taken to avoid confusing this with a pleural friction rub of pleuritis near the heart. The diagnostic criteria may be found in any standard textbook on physical diagnosis.

7. **Diastolic Murmur.**—A diastolic murmur indicates heart disease. The principal exception to this rule is a basal diastolic murmur which occurs during the last trimester of pregnancy. The murmur disappears dramatically upon delivery. When doubt exists about a basal diastolic murmur in a patient during pregnancy, it is advisable to reserve a definite opinion until after delivery. *Systolic murmurs are not reliable signs* of heart disease.

8. **Thrills.**—Turbulence of flow in the heart which is sufficiently great to be palpated as a *thrill*, whether it be systolic or diastolic in time, is a definite sign of heart disease. It must be a definite thrill or purr and not a vibration of the anterior thoracic wall from vigorous heart action, such as might occur in a patient with a thin chest and tachycardia.

9. **Generalized Senile Arteriosclerosis.**—Any patient with generalized arteriosclerosis must have coronary sclerosis, at least by inference. Although such a patient may have no cardiac abnormalities otherwise, he should be considered clinically to have cardiac disease and should be treated accordingly. Such management would certainly prolong the lives of many elderly people who resent aging and fail to do so gracefully. It is necessary that they resign themselves to the necessity of reducing their activity in accordance with their cardiac capacity.

10. **Thyrotoxicosis (Graves' Disease).**—Severe hyperthyroidism of several months' duration should be considered as having produced heart disease. There are some clinicians who feel that a normal heart will not become diseased from uncomplicated thyrotoxicosis. Experience has shown, however, that there usually is cardiac disease in these patients. With early and adequate treatment, this type of disease is reversible.

11. **Myxedema.**—Myxedema which is definite and accompanied by the classical clinical manifestations must be associated with cardiac disease. This type of disease, of course, is easily reversible if treated early.

12. **Classical Heberden Pain.**—*True or classical Heberden type of pain* such as seen in classical *angina pectoris* is definite evidence of heart disease. This pain is squeezing or vise-like, located over the precordium and referred to the epigastrium and along the ulnar side of the left arm into the fourth and fifth fingers. It may be referred to the left shoulder or left side of the head and neck. The pain occurs on exertion and is relieved by rest.

level The vein is brought to the phlebostatic axis or phlebostatic level[3] The needle (24 to 26 gauge) is inserted into the vein Once the needle enters the vein the venous pressure begins to force blood into the needle and the meniscus of the citrate solution in the adapter begins to move away from the vein The pressure in the system is then increased by means of the screw and pressure bulb in order to increase the air pressure against the meniscus The pressure in the phlebomanometer is increased until the meniscus is brought to a standstill At that moment the pressure in the pneumatic system of the phlebomanometer is equal to the pressure in the veins or venous pressure The pressure in the manometer is recorded as the venous pressure Since the pressure is read with the meniscus at a standstill small needles can be used with absolute accuracy

The method is simple The manometer can be placed anywhere that is on a table near the patient's bed on a shelf above the patient or at any convenient place The operation is no more difficult than an *intradermal* injection since the adapter and needle alone need be manipulated like any tuberculin syringe Because the needle is small it is possible to make measurements in small veins where pressure values are often most desired that ... in any
... Deh
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... —Diastolic hypertension for prolonged periods of time (eighteen months or more) indicates heart disease It is unlikely that a person who persistently has a diastolic blood pressure over 100 mm mercury will have a normal heart Not only does the hypertension itself produce strain on the left ventricle but the arteriolar disease renal dysfunction and other *associated* disturbances impair myocardial function

1) **Severe Anemia** Anemia with less than 50 per cent circulating hemoglobin of several weeks duration indicates heart disease This results in impairment of myocardial metabolism and hemodynamic function with increased cardiac work

II **Protodiastolic Gallop Rhythm** Definite protodiastolic gallop rhythm indicates weakness of the ventricular muscle It ... tain that one is not concerned merely ...

... clinical manifestation ...

mally, the veins *of* the dorsal surface of the hand which is on the thigh are collapsed and those that are on the dorsal surface of the hand on the table are distended. If the veins of both hands are distended, there is *venous hypertension*, if the veins of both are collapsed, there is *venous hypotension*

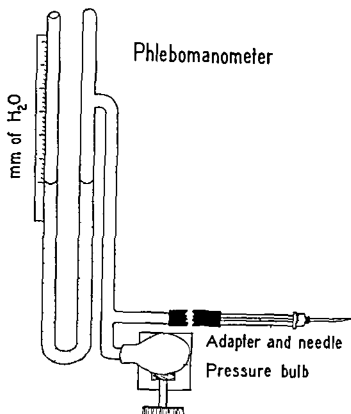


FIG 34 A diagram of the *Phlebomanometer* For recording a venous pressure the vein is brought to the phlebostatic level and passively held there A column of sterile 2 per cent sodium citrate solution is drawn into the distal third or half of the glass adapter *by creating a negative pressure in the system with the screw and pressure bulb* The pressure in the system is then readjusted to zero The needle is inserted into the vein being studied and the pressure within the vein slowly forces blood into the adapter *thereby moving the meniscus of the sodium citrate outward* *The* pressure within the system is slowly increased until the meniscus comes to a standstill At this time the pressure within the Phlebomanometer is equal to the pressure within the vein The pressure *in millimeters of water in the manometer is recorded as the* venous pressure

2 *Direct Phlebomanometry* The phlebomanometer (Fig 34) is a simple accurate and *direct method* for measuring venous pressure A column of sterile 2 per cent sodium citrate solution in water is drawn about half way up the adapter by creating a negative pressure with the pressure bulb The pressure in the system is then brought to atmospheric pressure zero

(e) **The Therapeutic Classification** is divided into five subdivisions indicating the degree of physical activity advisable

Class A Patients with a cardiac disorder whose ordinary physical activity requires no restriction

Class B Patients with a cardiac disorder whose ordinary physical activity needs no restriction but who should be advised

Class C whose ordinary physical activity should ... tricted and whose more strenuous habitual efforts should be discontinued

Class D Patients with a cardiac disorder whose ordinary physical activity should be extremely restricted

Class E Patients with a cardiac disorder who should be at complete rest or confined to bed

In order to illustrate the manner in which a complete diagnosis of heart disease is made let us consider a patient who had acute rheumatic fever several years ago and who now at the age of thirty years has auricular fibrillation and premature beats with mitral stenosis a large left atrium right ... lly ... the

auricular fibrillation ... complete diagnosis would be written as follows

I HEART DISEASE

A *Etiologic* Rheumatic fever
B *Anatomic* Mitral stenosis
Mitral insufficiency
Left auricular enlargement
Right ventricular enlargement
C *Physiologic* Auricular fibrillation
Ventricular premature contractions
Mitral valvular stenosis
Mitral valvular incompetence
Cardiac insufficiency (congestive) right and left ventricular
D *Functional* Class IV
E *Therapeutic* Class E

Once ... has been ... to be ... such a diagnosis can immediately obtain a fairly complete evaluation of the clinical state of the patient Such a diagnosis can be intelligently interpreted anywhere in the world It is defi... making ... should avoid the habit of ... has the ... stating that the patient ... heart failure,' an all too

These may be found in monographs on electrocardiography and will not be discussed here

DIAGNOSIS OF HEART DISEASE

When one or more of the *pathognomonic* signs of heart disease has been elicited a diagnosis of *heart disease* can be made After making a diagnosis of heart disease, the physician is obligated to make at least five additional diagnoses

(*a*) **Etiologic** —The attending physician must find the cause of the heart disease if he intends to institute the proper type of therapy In the treatment of any type of heart disease the removal of the cause or causes when possible is of paramount importance Many forms of heart disease are reversible when the cause is removable Furthermore a knowledge of the etiologic factor makes it possible to render a more accurate prognosis Consult the *Appendix* for a list of the etiologic factors concerned with heart disease

(*b*) **Anatomic** —An anatomic diagnosis must be made It is essential to indicate in the diagnosis the morphologic or structural changes that have occurred as a result of the action of the etiologic agents These structural changes should be definite clear and concisely written This diagnosis makes it possible to visualize and correlate properly the structural changes associated with the functional disturbances of the diseased heart (see Appendix)

(*c*) **Physiologic** —The physiologic state of the heart should be known The manner in which the cardiac physiologic state has been disturbed by the cardiac disease should be evaluated The observer should consider (1) the *nature of the cardiac mechanism* (2) *the functional state of the myocardium*, that is whether or not failure exists and (3) the disturbance in hemodynamics or the flow of blood (see Appendix)

(*d*) **Functional** —The functional capacity of the heart should be surveyed and recorded It is impossible to evaluate the cardiac reserve quantitatively The most accurate practical classification is as follows

Class I Any patient who is able to carry on his normal activity or that which any average normal person of his type should be expected to perform is said to be in *functional Class I*

Class II Any patient in whom moderate or extreme exertion brings on cardiac symptoms such as dyspnea palpitation weakness or cardiac pain is considered to be in *functional Class II*

Class III A patient is in *functional Class III* if he can perform the bare necessities of life such as dressing eating or walking slowly around the house without experiencing any cardiac discomfort

Class IV Any patient who is free from cardiac symptoms only when resting in [illegible]
Patients
function

The Backward Failure Concept With failure of the ventricle to pump blood onward it accumulates proximally in the atrium and veins This results in an increase in venous pressure impairment of blood flow and venous stasis a dam in the stream With the increase in hydrostatic pressure there is loss of plasma water and electrolytes into the interstitial spaces with formation of edema Furthermore because of the venous stasis anoxia of the capillary endothelium develops with an associated increase in capillary permeability and more edema These factors produce generalized edema pleural fluid and ascites engorgement of the hepatic sinusoids and hepatomegaly and generalized venous hypertension with engorgement of the veins of the neck

The Forward Failure Concept With failure of the ventricles there is insufficient supply of blood to the tissues and resultant anoxia of the capillary endothelium leading to an increase in capillary permeability Formation of edema ensues Impairment of blood flow to the tissues and a decline in *vis a tergo* produces stasis of blood in the peripheral blood vessels and further anoxia

There were some observers who considered congestive heart failure to be the result of both factors i e backward and forward failure The con-

[illegible]

[illegible] out that the mechanism of the syndrome was not established They pointed out certain incompatibilities especially in the backward failure concept some of which include

(1) Venous pressure has been shown to be extremely elevated in the absence of any significant amount of edema for example over 400 mm of water pressure in the legs following ligation of the inferior vena cava cardiac tamponade due to pericardial effusion or concretio cordis with venous hypertension without edema

(2) Many anoxic states such as occur with congenital heart disease pulmonary disease or high altitude are known to exist in the absence of edema or the syndrome of congestive heart failure

(3) *Cardiac output* a factor difficult to eval[illegible]

[illegible]

[illegible] disease and arteriovenous fistula will have a reduction of his cardiac output from the previously extra high level to a lower level when he develops congestive failure Although the lower level is too low to meet the circulatory needs of the tissues and the associated disease state so that the syndrome of congestive heart failure develops because of relative circulatory insufficiency, the new abnormally low level of cardiac output is still within the normal range

common practice Such a diagnosis is vague and carries no evaluation of the anatomic change cardiac mechanism severity, functional state or therapeutic class Remember there is only one satisfactory cardiac diagnosis and that is one which is *complete and detailed* as previously outlined The physician must visualize the cardiac status completely thus forming a mental picture of the anatomic and physiologic disturbances present He should form a mental *projection of the histologic changes* disturbances in blood flow that are occurring within the heart and vascular system in general as well as other alterations in the cardiac state

HEART FAILURE

Prior to a discussion of cardiac symptomatology the criteria for diagnosis and the classification of heart failure will be briefly presented

Heart failure is divided into two clinical types

1 Anginal
2 Congestive

1 **Anginal Failure** is exemplified by (a) *angina pectoris* and (b) *coronary occlusion* with myocardial infarction These have already been mentioned and will be discussed in more detail later This type of failure is often referred to as *coronary failure* or *coronary insufficiency* or a failure to supply an adequate amount of blood to the myocardium

2 **Congestive Heart Failure** —This is produced by failure of the myocardium to perform its main function as a pump It is synonymous with *myocardial insufficiency*

Congestive heart failure may be

A Left ventricular
B Right ventricular or
C Combined left and right ventricular

The physiology of heart failure or the several theories advanced today to explain the mechanism will not be discussed here It will be assumed that the reader possesses this necessary information before he progresses *further with this discussion*

MECHANISM OF CONGESTIVE HEART FAILURE

Because congestive heart failure is such a common clinical syndrome and

the mechanism of chronic congestive heart failure was clearly understood it is now realized that it is not known Congestive heart failure is an extremely complex physiologic state limited almost entirely if not exclusively to man It is under intensive study today

Up to a few years ago there were advanced two *classical* concepts of the mechanism of congestive heart failure They were *essentially as follows*

The Backward Failure Concept With failure of the ventricle to pump blood onward it accumulates proximally in the atrium and veins. This results in an increase in venous pressure impairment of blood flow and venous stasis a dam in the stream. With the increase in hydrostatic pressure there is loss of plasma water and electrolytes into the interstitial spaces with formation of edema. Furthermore because of the venous stasis anoxia of the capillary endothelium develops with an associated increase in capillary permeability and more edema. These factors produce generalized edema pleural fluid and ascites engorgement of the hepatic sinusoids and hepatomegaly and generalized venous hypertension with engorgement of the veins of the neck

The Forward Failure Concept With failure of the ventricles there is insufficient supply of blood to the tissues and resultant anoxia of the capillary endothelium leading to an increase in capillary permeability. Formation of edema ensues. Impairment of blood flow to the tissues and a decline in *vis a tergo* produces stasis of blood in the peripheral blood vessels and further anoxia

There were some observers who considered congestive heart failure to be the result of both factors i.e. backward and forward failure. The consensus was in favor of the Backward Failure Concept

Although the backward failure mechanism was formerly generally accepted and still is by some investigators today there were others who were of the opinion that the mechanism of the syndrome was not established. They pointed out certain incompatibilities especially in the backward failure concept some of which include

(1) Venous pressure has been shown to be extremely elevated in the absence of any significant amount of edema for example over 600 mm of water pressure in the legs following ligation of the inferior vena cava cardiac tamponade due to pericardial effusion or concretio cordis with venous hypertension without edema

(2) Many anoxic states such as occur with congenital heart disease pulmonary disease or high altitude are known to exist in the absence of edema or the syndrome of congestive heart failure

(3) *Cardiac output* a factor difficult to evaluate ...

... disease and arteriovenous fistula will have a reduction of his cardiac output from the previously extra high level to a lower level when he develops congestive ...
Although the lower level ...
tissues and the associate...
heart failure develop...
new abnormally low level of cardiac output is still within the normal ... circulatory insufficiency, the range

With *low output* failure the cardiac disease is associated with or due to disease states which do not necessarily modify cardiac output until the heart fails With cardiac failure the output of the heart declines from a normal level to a *low one* This new level is below the normal range and results in circulatory insufficiency and the syndrome of congestive heart failure

Whether or not these concepts of low and high output failure actually explain the physiologic observations remains to be fully established It is true however, that patients in heart failure may have a cardiac output

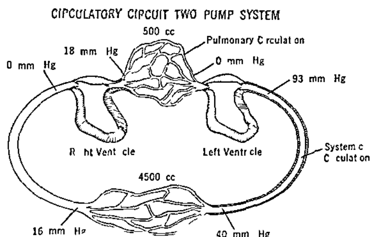


Fig 35 —Courtesy Am Heart J **41** 92 1951

within normal range Because of considerable discrepancies in measurement of cardiac output due to inaccuracies in the method of measurement differences in stages of the disease and therapeutic factors active within the patient at the time the role of cardiac output in the syndrome of chronic *congestive failure is not clearly established*

Furthermore therapeutic procedures not known to improve cardiac output will improve the clinical syndrome of congestive heart failure for example mercurial diuretics and restriction of sodium intake Again cardiac output has been observed to remain unchanged or actually to decline when the patient is improving from his failure

(4) The *edema fluid* is essentially an ultra filtrate of the plasma with about 0 5 Gm *proteins per 100 cc If capillary permeability were increased* is conjectured in the backward or forward failure concepts there should be an increase in the protein content

(5) Again almost the complete syndrome of failure is produced by administration of *desoxycorticosterone acetate* which is not known to have significant cardiac action

These and other observed phenomena have cast doubt upon the backward failure concept or the "dam in the stream" concept and have resulted in reconsideration of the mechanism of congestive heart failure

From the *hemodynamic* point of view, the dam in the stream mechanism for the increase in venous pressure and the syndrome does not seem tenable Details of the arguments invalidating a dam in the stream concept may be found in the medical literature These arguments may be summarized briefly as follows

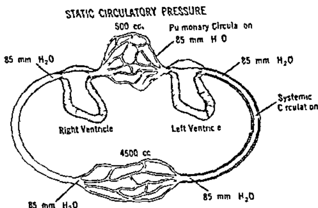


Fig 36 —Courtesy Am Heart J 41 928 1951

The circulatory system may be represented as a circuit containing two pumps right and left ventricles separated by the pulmonary and systemic circulations (Fig 35) Normally the two ventricles pump an equal volume of blood If for example each ventricle ejects 60 cc per stroke and they pump normally they will maintain the pressure gradient and distribution of volume of blood around the circulatory circuit as indicated by Fig 35 If both ventricles or pumps failed simultaneously and completely the circulation would stop and the pressure throughout the circulatory circuit would become static at about 85 mm H_2O (Fig 36) the *mean systemic pressure* of Weber The blood volume is only slightly redistributed a relatively s[illegible]

[illegible]

become reestablished

Should the *right ventricle fail* and pump less blood for example 59 cc per stroke while the left ventricle continues to function normally pumping 60 cc per beat more blood (1 cc per stroke) will be removed from the pulmonary system by the left ventricle than is pumped into it by the right one After a few minutes or relatively few beats 5 minutes with a cardiac rate of 100 per minute if this differential could be maintained the pulmonary circuit would be emptied of its 500 cc of blood and no more blood would be removed from the pulmonary system by the left ventricle than is

delivered to the pulmonary system by the right, *i e*, the left ventricular stroke volume would decline to that of the right ventricle 59 cc Blood would have been shifted from the pulmonary circuit into the systemic one If the entire 500 cc in the pulmonary system were shifted into the systemic portion of the circulatory circuit venous pressure would not be expected to increase on that basis alone provided the venous system were functioning normally It is well known that man can receive 500 cc or more of blood in transfusion *with little or no increase in venous pressure This blood*, incidentally, is shifted to the venous portion of the systemic vessels which is the large reservoir for blood in the body Therefore the dam in the stream cannot in itself be responsible for the increase in venous pressure seen in congestive heart failure That the venous pressure is increased with *failure of the right ventricle is established and, therefore must be explained* by some mechanism other than merely failure of the right ventricle to pump blood into the pulmonary vessels

The only possible mechanisms for the increase in venous pressure would then be either or both

(1) *Increase in blood volume in the veins*
(2) Increase in venous tone or tightness with which the veins squeeze upon the blood within

Since congestive heart failure may be associated with little or no change in blood volume, the venous tone must therefore be responsible for the *increase in venous pressure That an increase in venous tone is primarily* responsible for the increase in venous pressure remains to be demonstrated experimentally even though this is logically so In fact by the same logic presented previously the increase in venous pressure that follows complete obstruction of the superior and inferior venæ cavæ is not due to a *dam in the stream but rather to an increase in venous tone*

Accumulation of edema fluid throughout the body in association with the increase in venous pressure cannot be due solely to an increase in hydrostatic pressure As already indicated with other hemodynamic and physiologic functions normal high venous pressure alone does not produce severe edema *Accumulation of water and electrolytes in chronic congestive* heart failure must be due to alteration in renal function this will be discussed later

Conditions are somewhat different for *failure of the left ventricle* If the left ventricle fails and begins to eject 59 cc per stroke while the right ventricle continues to pump a normal amount (60 cc) per stroke then 1 cc of blood would be shifted from the systemic venous side of the circulation and placed in the pulmonary system If the heart rate is 100 per minute 500 cc would accumulate in the *pulmonary system every 5 minutes* Because there is a relatively large venous reservoir from which to draw the pulmonary system could become congested with blood with resultant acute pulmonary edema and dyspnea acute left ventricular failure Thus a dam in the stream on the left side of the heart can produce congestive failure *This is seen with mitral stenosis a mechanical dam* or obstruction to blood flow

The rate with which the blood would be shifted with either right or left ventricular failure would be determined by the heart rate and the degree of difference between the stroke volume of the two ventricles. It is evident that a small fraction of a cubic centimeter difference could produce serious difficulty in a relatively short time and still no existing method available today for measuring stroke volume of the ventricles could detect the difference, a serious shortcoming in the study of the pumping functions of the heart.

The *kidneys* play an important role in the development of the syndrome of chronic congestive heart failure. The evidence for this is considerable. It is known that the electrolytes and water accumulated in congestive heart fail ... entered the ... act and ... vidence ... edema fluid must have developed because of reduction in renal output. But why and how the kidneys fail to excrete the electrolytes and water remain unknown. The published data vary considerably because of variations in the degr...

... the kidneys to ... degree than ... ter. This is pro... not due to primary renal disease or dysfunction because during severe and progressing failure the kidneys will excrete subst...

... ...ver during congestive heart failure. But no definite chemical factor has been shown to be responsible for the *altered* renal function.

The medical literature should be consulted for details of the researches on renal function in congestive heart failure as well as other aspects of this complex and still unsolved clinical syndrome. Although the mechanism of chronic congestive heart failure is not clear, it appears that the syndrome develops in the following general manner.

First there is failure of the heart as a pump. When the heart fails to circulate an adequate amount of blood to the tissues for a fairly long period of time, there is set into motion a chain of event...

(1) ...
and th...
incre...
known ... for these changes remains un...

(2) ...
in the
...

settled. Chemical as well as physical factors such as hydrostatic pressure are involved in retention of the water and electrolytes.

(3) The renal function is altered with a resultant reduction in its excretion of water and electrolytes and other substances found in the urine, thus contributing this significant part to the electrolyte and water retention. The renal change is probably the result of hemodynamic changes such as those which produce a reduction in the rate of glomerular filtration and the result of chemical changes which alter renal tubular function. Hormonal factors of either or both adrenal and pituitary gland origin may be responsible in large part for the chemical alterations, but this remains to be established. The chemical factors responsible for the altered renal function as well as other changes in metabolism must have varied origins and complex integrations.

It is evident that existing knowledge of the mechanism of congestive heart failure contains numerous gaps. The mechanism for the integration of the many factors await elucidation; even the "trigger" mechanism responsible for the initiation of the syndrome is not known.

Regardless of the mechanism of chronic congestive heart failure, the clinician through many years of experience has learned that from the *therapeutic and clinical points of view it is easier to consider right and left ventricular congestive failure* in his patients with heart disease. Criteria for the diagnosis of failure of each or both ventricles must be known and elicited *in his patient. Even though the mechanisms are obscure, and* whether or not each ventricle can fail separately, he knows that when there exist certain types and degrees of clinical manifestations considered as evidence of ventricular failure, administration of specific procedures will achieve the best therapeutic results. This may be considered an empiric approach, but it remains the best today even though it is not the preferred one. Therefore in the discussions to follow the most practical present-day clinical approach is adhered to when necessary.

CRITERIA FOR THE DIAGNOSIS OF LEFT AND RIGHT CONGESTIVE HEART FAILURE

A. **Left Ventricular Congestive Heart Failure** is associated with any or all the following signs which are of diagnostic importance:

1. Cardiac dyspnea
   a. Dyspnea on exertion
   b. Orthopnea
   c. Paroxysmal nocturnal dyspnea
   d. Acute cardiac dyspnea or cardiac asthma
   e. Cheyne-Stokes respiration

2 Decrease in vital capacity
3 Accentuated pulmonic second sound
4 Roentgenographic abnormalities
5 Fine moist rales at the bases of both lungs
6 Protodiastolic gallop rhythm
7 Pulsus alternans

1 Cardiac Dyspnea — True cardiac dyspnea is a good sign of left ventricular failure The physician however makes certain that the patient has true *cardiac dyspnea* Whenever a hasty history is taken and a patient is simply asked if he has shortness of breath even normal persons are likely to say they do This is a common complaint among patients On the other hand *true cardiac dyspnea is found only in the patient with heart disease*

The average patient with shortness of breath when questioned closely, may be found to have sighing respiration a reliable sign of psychoneurosis *Sighing respiration* is exceedingly important in clinical medicine because of its significance frequency and because it is so frequently a source of confusion in diagnosis A person who complains of sighing respiration finds that breathing is comfortable but periodically there is the subjective impression that the lungs cannot be filled sufficiently with the associated sensation of suffocation resulting in a deep inspiration He will take several deep sighing breaths in an attempt to relieve the feeling that the lungs must be more completely filled If and when an impression of successful filling has been obtained the sense of suffocation disappears and comfort is obtained for a time varying from a few minutes to several days or even weeks whereupon this same experience is repeated At times this leads to a sensation of panic especially when the patient is already emotionally upset Since psychoneurosis is present anyway panic is prone to develop The episodes are not related to exercise and usually come on while the patient is quiet and morose or [illegible]

a Dyspnea on Exertion
exertion and in its early sta[illegible]
breathing with a sense of brea[illegible] ness and suffocation The patient tends to pant or breathe as though he has just run a race In severe cases the slightest exertion will precipitate dyspnea It must be remembered that pulmonary disease and other states produce the same type of dyspnea As the disease progresses dyspnea is present even at rest and is so severe that the patient has to sit up in bed or against a back rest [illegible]

[illegible]

[illegible] ate

1 *There is greater freedom of movement of the ribs and muscles of respiration* when the patient is erect The pump handle movement of the thoracic cage takes place more freely in the erect position than in the supine one

2 *The abdominal viscera gravitate to the pelvis* away from the diaphragm allowing freedom of movement of this structure with resultant improvement of the diaphragmatic element of respiration
3 *Pulmonary edema fluid gravitates to the base of the lungs* thus reducing the number of alveoli that are lined with a film of fluid Instead of having a large alveolar surface coated with a film of fluid which greatly interferes with O_2 and CO_2 exchange this fluid gravitates to a relatively small number of alveoli thus interfering less with total gaseous exchange
4 *Venous drainage from the respiratory center* in the medulla is improved thus eliminating venous stasis in this vital center and permitting more complete removal of metabolites which interfere with normal respiratory function
5 *Impairment of venous return from the lower part of the body reduces* the engorgement of the venae cavae and right atrium and consequently the load on the right side of the heart and probably in turn on the left ventricle In brief the load on the heart is decreased
6 *Hering Breuer reflex* is probably less exaggerated since the disturbances in pulmonary elasticity or distensibility are limited to a relatively small portion of the lungs that is at the bases where the fluid gravitates
7 *A decrease in cerebrospinal fluid pressure* in the brain and medullary *centers including the respiratory center follows when the patient* assumes the erect or sitting position Increased pressure on the respiratory center may produce dyspnea

c *Paroxysmal Nocturnal Dyspnea* is a good sign of acute left ventricular congestive heart failure The mechanism for this type of dyspnea is unknown Many ideas have been advanced but *none satisfactorily* explains the entire clinical picture (Fig 37) Most clinicians wisely refrain from offering a definite explanation An interesting concept sometimes advanced is as follows The left ventricle which is under a strain throughout the day as in hypertension aortic regurgitation or impaired coronary flow to the left ventricle recuperates more slowly than the right ventricle *when the patient rests in bed and falls asleep The right ventricle becomes* completely rested first and pumps more blood into the pulmonary circuit than the still fatigued left ventricle can remove This results in congestion of the pulmonary vascular system with acute dyspnea The patient suddenly awakens is extremely dyspneic and severely apprehensive especially if this is the first experience He suddenly sits up for relief or may run to an open window or a porch for fresh air The accompanying fear panic running and struggling increase the *demands made on the left ventricle*

[illegible] both ventricles and since the right [illegible] congestion of blood in the lungs is [illegible] and the patient becomes more frightened and fears that death is near A vicious cycle is established This type of dyspnea develops within an hour or two after the patient retires for the night or shortly after he falls asleep

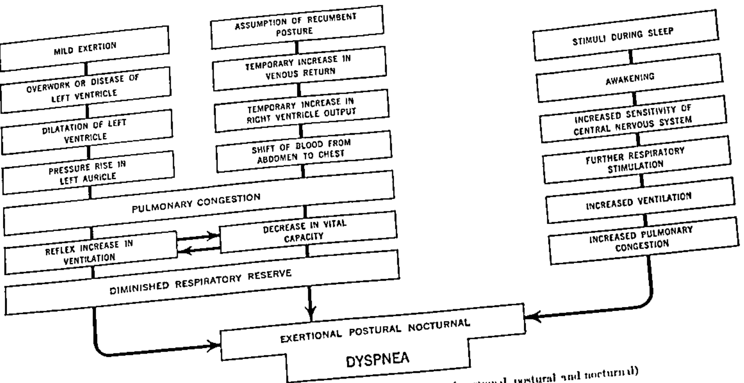


Fig. 37.—Diagram of the mechanisms involved in cardiac dyspnea (exertional, postural and nocturnal)

At times the *paroxysmal nocturnal dyspnea* does not appear until the night's sleep is about over that is about 4 or 5 o'clock in the morning. The mechanisms involved are essentially the same as those for the episodes that occur early during sleep. The accumulation of metabolites in the respiratory center produced by the sluggish circulation during sleep plus the added influence of an exciting dream which may precipitate acute failure of the left ventricle further aid in establishing the nocturnal dyspnea. These ideas of mechanism are illustrated diagrammatically in figure 37.

d. Acute Cardiac Dyspnea associated with wheezing or asthmatic breath sounds is often referred to as *cardiac asthma*. Any of the acute episodes of cardiac dyspnea of severe left ventricular failure may be associated with



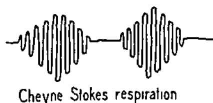


Fig. 38. Illustration of Biot and Cheyne-Stokes types of breathing. The periods of apnea and hyperpnea are apparent.

severe coughing, frothy sputum which is often bloody and gurgling râles. This is part of the picture of the acute pulmonary edema produced by acute left ventricular congestive heart failure.

e. Cheyne-Stokes breathing is a special type of periodic respiration which is encountered in many clinical states of which congestive heart failure is but one. The mechanism is not known but the explanation usually given is: Because of heart failure there is impairment of circulation to the respiratory center with anoxia and depression of the center. With depression of respiration metabolites such as CO_2, lactic acid etc. accumulate to fairly high levels. Once a sufficient concentration of these chemicals is reached *a strong stimulation of respiration results*. The respiration rapidly increases in depth and rate, the arterial blood is well oxygenated and the venous blood is cleared of excess CO_2. When this occurs the stimulus to respiration is reduced, respiration then decreases in rate and volume with a period of apnea finally setting in. The hyperpnea and apnea alternate in cycles (Fig. 38).

At times the cyclic respiration may be of the Biot type (Fig. 38) i.e. instead of a gradual change from the period of hyperpnea to apnea and *vice versa* the changes are abrupt.

2 A *decrease in vital capacity* (Fig 39) accompanies left ventricular failure but it is well to remember that it may occur with many types of pulmonary and other diseases as well A decrease in vital capacity aids in completing the picture of left ventricular failure and should be measured not only to make a diagnosis of failure but also to follow its progress

TABLE 1 —NORMAL VITAL CAPACITY

	Men	Women	Athlete
cc per cm height	25 0	20 0	29 0
cc per m² body surface	2500	2000	2800

3 *Accentuated Pulmonic Second Sound* P_2 —With failure of the left ventricle there is an accumulation of blood within the pulmonary veins and arteries This results in pulmonary hypertension with a greater force which suddenly snaps the pulmonary cusps closed when the right ventricle goes into diastole This sudden closure of the pulmonary cusps results in an accentuation of P_2 the pulmonary second sound It should be pointed

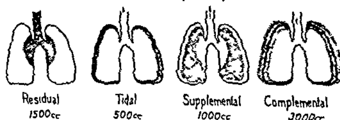


Fig 39 The [illegible]

out at this moment that in the younger age group up to or through adolescence the second pulmonic sound P_2 is louder than the second aortic sound A_2 As one grows *older* A_2 becomes louder and P_2 becomes relatively less intense With senility and senile emphysema P_2 again increases in intensity The intensity of P_2 is evaluated by its absolute loudness and especially by comparing [illegible]

[illegible] there is an [illegible] in the lungs and homogenous opacity in the lower portion of the pulmonary fields with pleural effusion

5 *Fine Moist Rales at the Bases Bilaterally* —With the accumulation of fluid in the parenchyma of the lungs with left ventricular failure there result fine moist (*crepitant*) râles These râles are more or less uniformly and equally distributed throughout the bases of the two lungs and are most evident posteriorly This sign is particularly important since *inflammatory disease of the lungs is not likely to be bilateral and symmetric* It is necessary that the observer rule out *marginal rales* which may be present in a patient confined to bed The latter type of rales usually disappear if the patient takes a few deep inspirations The rales of heart failure persist

6 *Protodiastolic Gallop Rhythm* is a good sign of failure of the left ventricle The mechanism, classification and diagnosis of gallop rhythm are discussed later (see Heart Sounds and Murmurs)

7 *Pulsus Alternans* is a pathognomonic sign of failure of the left ventricle Consult the foregoing discussion for the details concerning pulsus alternans

B **Right Ventricular Congestive Heart Failure** —Right ventricular heart failure is usually associated with the following manifestations which as a group constitute a diagnostic syndrome no one of them being diagnostic alone

1 Generalized and symmetric venous hypertension
2 Increase in blood volume
3 Dependent edema
4 Hepatomegaly
5 Pleural effusion and ascites
6 Anasarca

1 *Venous Hypertension* —The venous pressure is proportionately elevated in all veins The methods of measurement and evaluation have already been described Distended neck veins with the patient *in the* sitting position is a useful sign of increased venous pressure but it must be employed with caution

2 *Increase in Blood Volume* This requires special apparatus and therefore is not practical for general clinical use It is possible that in the future sufficiently accurate and simple methods will be developed for clinical use

3 *Dependent Edema* —Bilateral and symmetric edema in the lower extremities is usually an early sign of failure of the right ventricle *The edema increases during the day as the patient is up and about and disappears with rest in bed* As the edema becomes more severe *it will subside only* with bed rest or not at all in the most severe instances of failure The edema becomes worse as the patient moves around during the day because the effect of gravity is to increase the hydrostatic pressure in the vessel of the feet and increase the force of the intracapillary filtration pressure When the patient reclines in bed the gravitational force is relieved Furthermore with rest in bed the demands on the heart and circulation are decreased the heart rests the cardiac reserve increases and the manifestations of heart failure subside There is much evidence to indicate that urine volume and electrolyte excretion are decreased when the subject is in the erect position and increased in the supine position The

intracellular tissue fluid is mobilized and excreted by the kidneys. The fluid and electrolyte intake also ceases at night. During the day, the demand on the heart is greatest; therefore, failure is most likely to be pronounced. Furthermore, electrolyte and fluids are being consumed.

4. *Hepatomegaly.*—Enlargement of the liver occurs early with failure of the right ventricle. The enlargement is due to congestion within the central veins and sinusoids brought about by the venous hypertension. This leads to distention of the hepatic sinusoids, edema, and swelling (cloudy swelling and hydropic degeneration) of the liver cells with resultant hepatomegaly. Enlargement of the liver leads to distention of Glisson's capsule with consequent spontaneous pain or tenderness. The liver in heart failure is diffusely and symmetrically enlarged. This enlargement may be detected on abdominal palpation and may be measured clinically by the distance which the lower edge of the liver is displaced below the costal margin in centimeters or fingerbreadths. Such a quantitation is grossly inaccurate but is exceedingly useful clinically in diagnosis, therapy, and in the evaluation of the course of the failure.

5. *Pleural Effusion and Ascites.*—The factors causing the cardiac edema in the legs or other intercellular spaces also result in an accumulation of fluid in the peritoneal cavity (ascites) and in the pleural spaces (hydrothorax or pleural effusion). This fluid is easily detected on physical examination. The fluid that accumulates in congestive heart failure in the pleural and peritoneal spaces is a *transudate* and not an *exudate*. The former is the result of fluid loss from the blood stream by a process of disturbances in the steady state of intra- and extravascular fluid exchange; the latter is a result of inflammation. Because of the differences between the two types of mechanisms, the fluids differ qualitatively (Table 2). Differentiation of these two types of fluids is exceedingly important clinically.

TABLE 2. DIFFERENCES BETWEEN EXUDATES AND TRANSUDATES

	Transudate	*Exudate*
1. Color	Serous, pale yellow	Turbid, purulent, hemorrhagic, chylous or a combination of these
2. Transparency	Clear	Clear or cloudy
3. Specific gravity	Less than 1.015	Greater than 1.018
4. Clotting	Does not clot	Clots spontaneously
5. Protein content	Less than 2.5 per cent	Greater than 3.0 per cent
6. Sugar content	Same as blood	Less than blood
7. Cell content	Few (mesothelial and erythrocytes)	Many (type depends upon nature and degree of inflammation)
8. Bacteria	None	Usually present
9. Seromucin (Rivalta Test)	Absent	Present

6. *Anasarca.*—When the generalize

5 *Fine Moist Râles at the Bases Bilaterally* —With the accumulation of fluid in the parenchyma of the lungs with left ventricular failure, there result fine moist (*crepitant*) râles These râles are more or less uniformly and equally distributed throughout the bases of the two lungs and are most evident posteriorly This sign is particularly important since inflammatory disease of the lungs is not likely to be bilateral and symmetric It is necessary that the observer rule out *marginal râles* which may be present in a patient confined to bed The latter type of râles usually disappear if the patient takes a few deep inspirations The râles of heart failure persist

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300 mm of water near the arteriolar end of the capillary and 390 mm near the venular end This slight difference is due to the escape of water from the capillary at the arteriolar end with slight concentration of the blood constituents as the blood passes along the capillary The returning

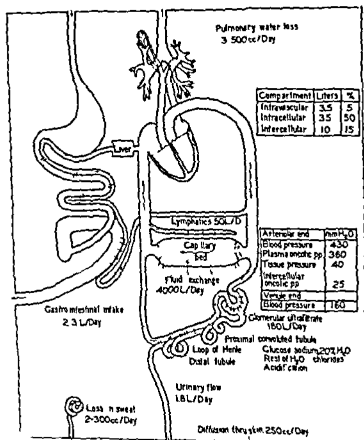


Fig 40 A diagrammatic presentation of the water compartments and exchange in a normal adult man under basal conditions in a comfortable environment The forces usually considered in the transfer of water across the capillary membrane are shown These are average values reduced to the [illegible]

pleural effusion and ascites the patient is said to have anasarca This may occur in severe and advanced right ventricular congestive heart failure There is usually associated left ventricular failure with edema of the lungs in [illegible]

[illegible] —When there is fai[illegible] previously for fai[illegible]

It is well to remember that any one sign with rare exception is not pathognomonic evidence of congestive failure It is the accumulation of all or most of them as a group which forms a syndrome diagnostic of failure of one or both ventricles In the study of a patient with heart disease this should be kept in mind When the clinician begins to consider the existence of congestive failure he should seek the signs of failure in a systematic manner To elicit one of the less reliable signs and then suddenly decide that congestive heart failure is present will lead to frequent serious and embarrassing errors in clinical diagnosis and treatment

INTRA- AND EXTRAVASCULAR FLUID BALANCE

There are a number of factors which are concerned with the exchange of extra- and intravascular fluids For practical purposes these can be considered to occur solely in the capillaries (Fig 40)

A **Forces Which Tend to Make Fluid Escape from Blood Vessels** — 1 *Intracapillary Blood Pressure (hydrostatic pressure)* The intracapillary blood pressure or hydrostatic pressure with the vessel at heart level is about 430 mm of water at the arteriolar end of the capillary and 160 mm at the venular end When the vessels are open and circulation is free these pressure levels are fairly well maintained This pressure tends to force fluid out of blood vessels Consequently an increase in this pressure will force greater quantities of fluid into the intercellular spaces

2 *Colloid Osmotic (Oncotic) Force of the Intercellular Fluid Proteins* — The colloid osmotic force exerted by the intercellular fluid proteins is approximately 25 mm of water at the arteriolar and venular ends Colloid osmotic pressure is due mainly to the albumin fraction because of the small size of this molecule Since the capillary wall is not freely permeable to the protein molecule a much higher concentration is maintained in the blood than in the tissue fluid Were it freely permeable the differences in protein concentration across the wall would not exist and no pressure gradient would be found The oncotic force of the plasma proteins almost equals the hydrostatic force of the capillary blood pressure In edema states associated with lymphatic obstruction there results an accumulation of proteins in the intercellular fluids with an increase in the osmotic force offered by the intercellular fluid It is well to remember that one of the principal functions of the lymphatics is to return proteins to the blood stream

B **Forces Which Cause Water to Return to the Blood Stream Across the Capillary Wall or Membrane** — 1 *Colloid osmotic (oncotic) force of the plasma proteins* The osmotic force offered by the plasma proteins is about

(1) the body as a whole and the environment and (2) the tissues and blood stream The ideas and explanations presented here include many of the generally accepted ones They are *grossly inadequate* Fortunately, the forces concerned with water exchange do not exist as generally presented If they did it would not ...
constantly edematous
and discussed today
tions exist Nevertheless because these theories are generally accepted

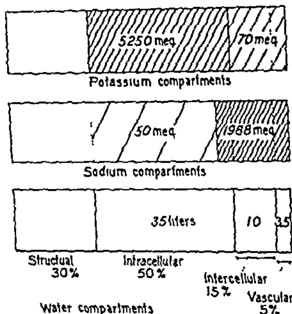


FIG 41 —A comp...

... tion of Na is about two-thirds ... concentration whereas the average urinary concentration of potassium is about ten times that of the blood The potassium is primarily intracellular Therefore there is a fairly rapid flow of potassium through the ... cellular spaces to reach the urine These ... states Further more the va... The value for the intracell...

concepts students are called upon to recite them and more important, they contain some truth Furthermore their inadequacies lead to thought Modifications have been made in order to approach more accurately the observed facts

Fluid Intake and Output —It is *obvious* ...

force of the plasma proteins is about equal to the hydrostatic force exerted by the intracapillary blood pressure

2 *Tissue pressure* is the pressure or force with which the tissues resist distention or displacement by fluid It averages about 38 mm of water but varies a great deal throughout the body (Table 3)

TABLE 3 TISSUE PRESSURE IN MM OF WATER FOR FOUR COMMON EDEMA SITES IN MAN

	Dorsum of Hand	*Volar Surface of Forearm*	*Pretibial Area*	*Dorsum of Foot*
Mean	18	21	37	31
Maximum	30	40	54	43
Minimum	8	11	18	15

C **Lymphatics** — Another important means by which fluid is returned to the circulation is by way of the *lymphatics* These structures play a variable and important role in maintaining normal hydration of the tissues The lymphatics are mainly responsible for the return to the blood stream of protein molecules that escape through the capillary wall into the tissue spaces It is estimated that over 90 per cent of the protein molecules that escape into the intercellular spaces are returned to the circulation by way of the lymphatics Some of the remaining molecules reenter directly into the capillaries as intact protein molecules and some are split into amino acids by enzymes Were it not for the lymphatics the oncotic force of the intercellular fluid would reach great heights and man would be edematous (lymphedema) at all times

D **Capillary Permeability** The degree of capillary permeability influences the state of tissue hydration because it influences the rate of escape of proteins into the intercellular spaces This additional amount of intercellular fluid protein influences the amount of fluid held outside of the blood vessels by oncotic force Capillary permeability is difficult if not almost impossible to measure It is concerned with spaces in the wall of the endothelial cells as well as the cement substance and the spaces between the endothelial cells

E **Electrolytes** It is generally believed that except in acute disturbances electrolytes crystalloids and water are freely diffusible and therefore are not concerned with osmotic forces Disturbances in the other factors lead to edema and in turn to shifts of electrolytes among the fluid compartments (Fig 41) of the body This may not be true however Studies with radiosodium have shown that at least 0 pounds of sodium chloride diffuse back and forth daily across the capillary walls of the normal man This great turnover makes it necessary to consider these massive shifts in electrolytes whenever edema is encountered The use of radioactive and stable isotopes as tracer elements may clarify the role of electrolytes The rôle of the kidneys still unclarified is important in regulating the state of hydration and electrolyte balance

EDEMA

A discussion of cardiac edema cannot be adequate without a brief review of the known mechanisms concerned with the exchange of fluid between

Mechanism of Diffuse Edema.—It is evident that in the usual patient with diffuse or generalized edema the mechanism can be focused to a large extent upon the kidneys, that is, there was at some time relative oliguria with intake exceeding output. It is unlikely and almost inconceivable that any person would develop edema from drinking too much fluid, being otherwise entirely normal. Thirst may exist or increase with certain edema states, a fact not impressive clinically, but in the presence of normal functioning kidneys it is unlikely to result in edema. Therefore, diffuse or generalized edema is one or a combination of factors that can be classified thus:

1. Renal
2. Prerenal
3. Postrenal

1. **Renal Edema.**—When the disturbance of water and electrolyte excretion (oliguria) resides primarily in the kidneys, the edema is *renal*. A good example of this type of edema is seen in acute mercurial poisoning. The tubular damage results in severe oliguria or anuria, so that the kidneys are unable to excrete water or other urinary constituents. Thus, except for water lost through the skin, lungs, and other extrarenal sites, all water and salt ingested or administered parenterally by the doctor must be *retained*. Therefore, the patient develops edema. This is primarily renal *in origin*. The same mechanism is true in part for *acute hemorrhagic nephritis* (Fig. 43) with glomerular damage. If no fluid were taken into the body, neither type of patient would develop edema. Cardiac edema (Fig. 42) can conceivably be mainly *renal edema*.

2. **Prerenal Edema.**—When disturbances in *formation* of urine result in oliguria or reduced urine volume and the fluid intake exceeds the output, not because the kidneys are *primarily at fault* but because the quantity and quality of the blood reaching the kidneys are reduced and altered respectively or because *of local* tissue or circulatory changes, prerenal edema develops. Thus the kidneys *have* an insufficient amount of water with which to urinate or bring into play normal mechanisms to conserve water and electrolytes, and *prerenal edema* follows. A good example of this type of edema is that seen with *acute lymphatic obstruction* (Fig. 45). The *obstruction of the lymphatics in the legs*, for example, results in edema although the kidneys would be capable of excreting the water retained. However, this fluid does not reach the normally functioning kidneys for excretion.

3. **Postrenal Edema.**—This type of edema is encountered whenever there is postrenal obstruction to urinary flow sufficiently great to produce oliguria or anuria. A *good example* of this is that seen in a patient with prostatism and complete urethral *obstruction*. The urine f[illegible]
be excreted [illegible]
uret[illegible]
of u[illegible]
fore[illegible]

him to the next over a *period of days the balance is steady*. *It must also* be admitted that whenever the intake of water exceeds the output *edema* to some degree must exist. The degree of edema is a quantitative index of the amount by which fluid intake exceeds fluid output. The most accurate and practical method for measuring the quantity of edema is to weigh the patient. In the average person *intake* of fluid occurs solely through the mouth and water is lost mainly in the urine. Therefore in simple term all *general edema states* can be considered to be an inadequacy of urinary volume so that fluid intake exceeds output. This must be true for cardiac, nephritic, nephrotic, lymphatic and other types of diffuse edema. Since water cannot be stored in the tissues in a hypotonic state, electrolytes must also be retained, that is, more electrolytes are ingested than are excreted in the urine. It is obvious that a discrepancy between the amount of fluid and electrolytes taken in and that lost in the urine may exist for only a *short time. Once the edematous state has developed*, a new balance between intake and output is established with the patient overhydrated. The problem then becomes one of explaining the mechanism by which less urine *is formed, for if the intake were to be voluntarily reduced by an* amount equal to the oliguria, there would be no gain in total body water.

It is well to diverge for a moment to point out that *edema is best defined as a greater than normal amount of intra- or extracellular fluid* and not necessarily an increase in total body water. The latter is usually true in generalized edema. That edema can occur without any gain in total body water is well known to everyone and can be well exemplified by the edema which occurs locally at the site of a bee sting. Fluid and electrolytes are shifted to the area of the sting from other portions of the body. It is also possible for interstitial fluid and electrolytes to exceed the normal amount with edema formation by a shift of water from the intracellular compartment. It must also be remembered that a person may be *physiologically dehydrated and still in a state of edema* and apparent overhydration. This would occur if large amounts of fluid shifted into the tissue spaces from the blood stream and intracellular compartments. Such states of functional dehydration can result in serious clinical disorders.

It is also well to remember that the edema fluid must be *qualitatively different* to some degree from the fluid normally present in that compartment. To date these qualitative differences have received insufficient study. The importance of these differences is acknowledged both physiologically and therapeutically.

When disease states are under consideration, such avenues of intake and output as intravenous and subcutaneous therapeutic administration, diarrhea, *vomiting, draining* wounds, fistula, etc., must be properly evaluated as to water and solute exchange. Many patients are made edematous by the doctor who forces water and solutes into the body parenterally, for example *intravenously*, when the volume of urine is reduced.

Even though the foregoing mechanisms are generally accepted concepts many observations have been made which cast doubt on their rôle in the development of the edema. As indicated previously, vena caval ligation often causes extreme elevations (600 or more mm. of water) of the venous pressure in the lower extremities without edema formation. Ordinarily, the edema level in terms of antecubital venous pressure is 150 mm. of water. Furthermore the protein content of edema fluid is low, rarely exceeding 0.5 gm. per 100 cc., thus tending to rule out the existence of increased capillary permeability. In addition the plasma proteins are normal or elevated but are rarely low in congestive heart failure. Therefore, in view

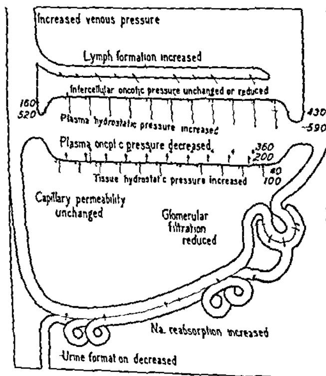


FIG. 42. In congestive heart failure the water exchange is ... figure 40 except that which ...

... there is also an in... Whether or not the latt... venous hypertension or t... water loss to the tissue spaces ... pressure in the ...

not become edematous if he ceased to take in fluid simultaneously with the development of the urinary obstruction With thought the student can classify the various edema states into any one or a combination of the foregoing three categories A detailed presentation of all the various forms of *edema will not be included here these special considerations will become* evident from the discussions which follow

In the discussions to follow concerning the factors and forces at work in the capillaries and other peripheral vessels it becomes immediately obvious *that the body as a whole and the kidneys in particular is involved in all* considerations of generalized or diffuse edema It is impossible to consider all factors or to understand any mechanisms in detail Edema formation is not well understood even in general terms It will be noted immediately that some of the factors *function as prerenal forces* whereas others are renal They must all be properly integrated Many are erroneous quantitatively and qualitatively as presented This unfortunately is the result of inadequate data An attempt has been made to present them here in the most acceptable terms of intra- and extravascular fluid balance The shortcomings in present day explanations will be obvious to the thouhgtful reader

I **Hormones** —There are many hormones many of which are unknown or only slightly understood which aid in regulating the state of hydration by both renal and extrarenal mechanisms Examples include the fluid and electrolyte regulating hormones of the suprarenal glands To date little is known about these hormones including their origin chemical structure and mode of action The role of antidiuretic and diuretic hormones is not understood in normal and edema states They offer a fruitful field for research

In the normal person the foregoing factors are all in a state of activity which maintains normal tissue hydration The forces involved are indi*cated in figure 10 It is obvious that if they alone were considered normal* man would be on the verge of edema at all times Simply lowering a part below heart level should produce edema It is known that this is not true for we see little or no edema of the feet in the normal person Therefore *other factors of considerable importance must control fluid exchange* between the blood vessels and tissue fluid The lymphatics kidneys muscles chemical and physical state of the inter and intracellular material etc must contribute

Specific Types of Edema — A Cardiac Edema In congestive heart failure with the development of *cardiac edema* there result (1) an increase in venous pressure and capillary *hydrostatic pressure* which elevates the filtration pressure (2) circulatory stasis in the peripheral blood vessels which results in *anoxia of the capillary epithelium* this contention has been made but it has not been shown to *increase capillary permeability* (3) a decrease in blood proteins because of anorexia dyspepsia and malnutrition resulting in a *decreased osmotic force* of the *plasma proteins* and (4) alteration in *renal excretion* which accompanies the failure (Fig 42)

example that starvation is often associated with plasma protein levels well below the edema level in the absence of any edema. Furthermore in many instances edema developed when the blood protein levels exceeded this critical concentration. The other disturbances in nutrition such as impaired intake of the vitamins, iron, calcium and other essential foods contribute to the edema by influencing capillary permeability and intra- and extracellular electrolyte balance. The renal factor must not be overlooked (Fig. 44).

Fig. 43.—A...
the same in...
occur in the...
gestive heart...
figures and...
of proteins ...
dicate the normal forces; the solid arrows the forces changed by nephritic edema.

Th... ...
I...
pre...
... the circulation. The accumulation of intercellular protein results in fibrin formation and later interstitial fibrosis and a woody or ligneous nonpitting edema. There is, therefore, a local disturbance in the effective colloidal osmotic force of the plasma and intercellular fluid proteins (Fig. 45).

of these inconsistencies a *more critical and skeptical interpretation of the* mechanism of congestive heart failure is required The problems of renal and hormonal dysfunction retention of sodium and water and increase in blood volume deserve further study

The role of the lymphatics with possible impairment of lymphatic contractions and failure of their valves has received little experimental consideration In addition to this the venous hypertension associated with congestive heart failure may play an important role in preventing adequate lymphatic drainage into the venous system In summary the *mechanism of cardiac edema has not been established* That more water and electrolytes are retained than excreted is definite The mechanism for this remains unsolved Reference to the discussion on the mechanism of congestive heart failure will reveal some of the existing concepts

B **Nephritic Edema** The generally accepted basic mechanism for the production of edema in acute hemorrhagic nephritis is an *increase in capillary permeability* This alleged increase purportedly allows plasma proteins to escape into the tissue spaces thereby increasing the osmotic pressure of the intercellular fluid and decreasing the osmotic pressure of the plasma This then results in a decreased effective plasma oncotic pressure with the hydrostatic force less opposed The outward diffusion of fluid is predominant and edema develops Such a mechanism is negated by the observation that the protein concentration of interstitial fluids in acute nephritis is not usually increased

The loss of proteins in the urine aids somewhat in the decrease in plasma proteins This is not of great importance in the average patient There is some evidence to suggest that in many patients congestive heart failure plays a prominent role in the formation of edema It is contended that the edema is firm or rubbery in character and does not pit as readily as other types of edema because of the high protein content of the tissue fluid (Fig 43) The edema is due in great part to reduced urinary excretion of water and sodium chloride as in congestive heart failure

C **Nephrotic Edema** Nephrotic edema has been said to result from an extreme reduction in plasma proteins with a proportionate fall in the colloid osmotic pressure of the plasma It is also contended that the total plasma proteins must fall below 5 5 gm per 100 cc of plasma or the albumin fraction to less than 2 5 gm per 100 cc of plasma before the colloid osmotic pressure of the plasma proteins will decline sufficiently to result in edema The albumin molecule being the smallest contributes most to the colloid osmotic forces and therefore a unit change in this fraction results in greater effects than a unit change in the other plasma protein fractions

Any clinical state which is associated with an extreme decrease in plasma proteins is supposed to result in a *nephrotic type of edema* *Nutritional edema* with low plasma proteins because of inadequate protein intake or poor assimilation results in nephrotic edema Recent studies however cast doubt on the generally accepted concepts concerned with nutritional edema and the associated low plasma proteins It has been found for

levels perceptible by ordinary clinical means it is said to be *latent* or *occult*. Edema is not evident clinically until the part has increased in water content by an amount equal to about *10 per cent of the weight of the part* involved. When an edematous area is pressed upon the fluid is displaced locally, leaving an indentation or pitted area thus pitting edema. Thin or less viscous edema fluid in loose tissue is easily displaced and pits readily, whereas viscous fluid in closely knit tissue pits with difficulty.

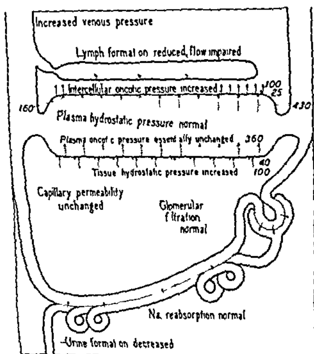


Fig. 45. In [illegible] the water and electrolyte forces throughout the body are essentially normal (Fig. 40). Th[illegible]

Tissue Pressure and the Skin.—The significance of the role of *tissue pressure* and the skin in formation of edema increases as the edema fluid increases and the hydrostatic pressure of the tissue fluids rises. The skin serves as a *limiting wall* which tends to prevent further accumulation of fluid and development of edema. [illegible] may be densely knit tissues [illegible] higher tissue pressure per unit [illegible] eyelids are more subject to [illegible] cumulation under lower pressures

E. **Inflammatory Edema.**—With inflammation there is local vasodilatation, venous stasis, increase in permeability, and lymphstasis with resultant edema.

F. **Thrombophlebitic Edema** —The venous obstruction in thrombophlebitis results in an increase in venous pressure in the tributaries not sufficient in itself to cause edema This must contribute to the formation of edema, however The inflammation of the veins extends to and involves the adja-

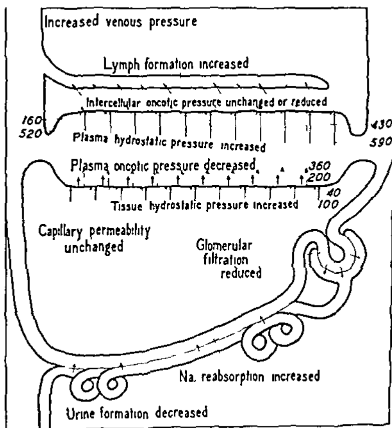


Fig 44 —The renal factor in *true nephrosis* is not too well known because this disease is so rare Most *nephrotic syndromes* are the result of chronic glomerular nephritis The low plasma proteins with decreased plasma oncotic pressure are believed to favor water loss into the tissues Severe sodium retention by the kidneys may be a significant factor, however (see text) The behavior of the electrolytes and water in the other portions of the body is much like that in the normal person (Fig 40) Consult the text for details The dotted arrows indicate the normal forces, the solid ones the forces changed by *nephrotic edema*

cent lymph channels, resulting in lymphangitis, lymphadenitis and lymphstasis. Impairment of lymph drainage contributes to formation of edema There also appears to be an arteriolar spasm which may enhance the formation of edema

General Considerations of Edema.—Edema may be defined as an abnormal amount of fluid in a tissue When this accumulation is below

Chapter 3

THE APPROACH TO A CLINICAL CARDIAC EVALUATION

THE patient with heart disease should be approached for a clinical survey and evaluation in a manner which should be employed for all patients regardless of their problem The study should always be complete careful and leisurely enough to permit the proper weighing of all data collected If this is not done the patient should be informed that the study has not been complete The circumstances should be explained so that he may decide whether or not he wishes to seek further study elsewhere All phases and methods of study should be employed An evaluation based on all methods available today is still inadequate and it is therefore imperative that no method or procedure be withheld from the study unless it is impossible to employ it No study can be too thorough or detailed In many instances unfortunately the studies are qualitatively and quantitatively poor

THE HISTORY

It is not the purpose of this monograph to discuss in detail the meth[illegible]

[illegible] the history when properly taken is the most important phase of the clinical study This obviously must include factors such as familial prevalence of heart disease past illnesses social circumstances habits nutrition and occupation

Certain symptoms represent common complaints of the patient with cardiac disturbances The three most frequently mentioned are

1 *Precordial pain*
2 *Dyspnea and*
3 *Palpitation*

1 **Pain** —Th[illegible]
who present [illegible]
sticking pain [illegible]
sis It may [illegible] it is not related to exercise or hard work and usually begins during periods of anxiety or emotional disturbance If the examiner carefully searches the region of this discomfort, he will almost invariably find a sharply localized [illegible]
tenderness [illegible]
type of pain [illegible]

In general, the factors and mechanisms concerned with formation of edema are little known *The metabolism of electrolytes, antidiuretic* diuretic and other hormones, and bound water are among many factors that seem to be important but receive relatively little consideration in clinical discussions today

It should be reiterated that all of the accumulated edema fluid water and solutes, enter the untreated patient by way of the gastrointestinal tract and most is excreted by the kidneys In brief, the patient with edema invariably has been in positive water and electrolyte balance at some time This positive balance is temporary and a new steady state of water and salt exchange both within the body and between the body and its environment is established when the patient becomes edematous Almost all studies concerned with the mechanism of edema formation fail to include the period of time during which the water and electrolyte accumulations *are being established and the factors initiating the edema* are at work Because the resultant edematous state alone is studied, many factors are not observed The rapid turnover of tremendous quantities of water and electrolytes must be considered in all forms of edema, particularly generalized edema

Chapter 3

THE APPROACH TO A CLINICAL CARDIAC EVALUATION

THE patient with heart disease should be approached for a clinical survey and evaluation in a manner which should be employed for all patients regardless of their problem. The study should always be complete, careful and leisurely enough to permit the proper weighing of all data collected. If this is not done, the patient should be informed that the study has not been complete. The circumstances should be explained so that he [illegible] else-where [illegible] evalua-tion ba[illegible] It is therefore imperative that no method or procedure be withheld from the study unless it is impossible to employ it. No study can be too thorough or detailed. In many instances, unfortunately, the studies are qualitatively and quantitatively poor.

THE HISTORY

It is not the purpose of this monograph to discuss in detail the method for taking an adequate clinical history. This should be common knowledge. Its application in cardiac disease is varied only insofar as the emphasis is on cardiac symptomatology and related problems. The history, when properly taken, is the most important phase of the clinical study. This obviously must include factors such as familial prevalence of heart disease, past illnesses, social circumstances, habits, nutrition and occupation.

Certain symptoms represent common complaints of the patient with cardiac disturbances. The three most frequently mentioned are:

1. *Precordial pain*
2. *Dyspnea*, and
3. *Palpitation*

1. **Pain.** The most common type of discomfort experienced by the patient who presents himself for a cardiac examination is a dull *ache or sharp sticking* pain over the precordium. This is most often due to psychoneurosis. It may be severe at times. It is not related to exercise or hard work and usually begins during periods of anxiety or emotional disturbance. If the examiner carefully searches the region of this discomfort, he will almost invariably find a sharply localized area of [illegible] tenderness. [illegible] type of pain

neurosis Its cause is unknown It is possibly a localized area of fibrositis and myositis and even periostitis brought on by increased muscular tone and activity so common in anxiety states The patient palpates the area so frequently that he often aggravates the soreness

The pains of *angina pectoris* and *myocardial infarction* have already been described

Acute pericarditis is often associated with cardiac pain This is probably due to inflammation of the pericardium and surrounding tissues adjoining the pleura and diaphragm It is usually a diffuse *precordial soreness* accentuated by pressure over the precordium and by respiration It may be referred to the shoulders (usually the left) when the diaphragm is involved

Acute myocarditis such as is encountered in acute rheumatic fever will often produce precordial *soreness* and pain The pain may be sharp and is aggravated by pressure over the heart

Acute aortitis such as that associated with syphilis will often produce a sense of oppression and discomfort in the substernal region near the base of the heart This pain is often difficult to distinguish in quality from that of myocardial infarction It may be referred to the neck and shoulder The pain is probably due to inflammation of the aorta with disturbances in the circulation through the *vasa vasorum* There may be infringement of the inflammatory process onto the region of the orifices of the coronaries with resultant impairment of the coronary circulation Reflex coronary spasm may be partially responsible for the pain The pain in the latter two circumstances follows *coronary insufficiency* and therefore simulates angina pectoris If an *aneurysm* of the aorta is present the pain may become severe and diffuse depending upon the location and extent of the dilatation

Severe anemia may produce a similar type of pain due most probably to myocardial anoxia

The pain of a *dissecting aneurysm* has much the character of myocardial infarction It is tearing in character severe and diffuse being felt over the chest and extending into the back arms neck and abdomen and often into the legs This pain like that in coronary occlusion is severe and persists for many hours to several days if death does not supervene

Severe cyanosis may produce a type of pain known as *angina hypercyanotica* It occurs in the presence of mitral stenosis if associated with severe cyanosis This pain is a localized oppression or heaviness in the precordium

The pain due to neuritis osteitis or pleuritis might be confusing and therefore deserves consideration during evaluation of the cardiac patient

2 **Dyspnea** —Dyspnea has already been discussed One of the early signs of left ventricular congestive heart failure is the presence of dyspnea As a result of this the patient often finds it necessary to sleep with several pillows in order to be comfortable *This is an important point to check when taking the history*

3 **Palpitation** Palpitation may be described as consciousness of the heart beat It is usually but not necessarily associated with tachycardia *Palpitation may* exist at normal heart rates or with premature contractions

The patient who has been told that he has heart disease or who suspects heart disease in himself is extremely conscious of his heart beat and often experiences palpitation In fact, he may find it almost unbearable to lie on his left side because he is overly conscious of his heart beating against the bed (*trepopnea*) *Patients with heart disease experience palpitation after* the slightest exertion when it should ordinarily only follow severe exercise Palpitation occurs early in congestive heart failure

The other symptoms commonly encountered in heart disease are

4 *Weakness*
5 *Dyspepsia*
6 *Cough and expectoration*
7 *Miscellaneous*

4 **Weakness** —Weakness is one of the most common complaints of the cardiac patient This symptom is rarely emphasized in the standard textbooks and monographs on heart disease It may vary from ease of fatigability to utter exhaustion in severe cardiac disease The degree of weakness is directly related to the reduction in the circulation to the tissues Weakness may be extremely prono need n the pro [illegible]

[illegible] of anorexia epigastric fullness nausea constipation pyrosis (heart burn) and gradual loss of weight It varies with the severity of the cardiac disease

6 **Cough and Expectoration** These are common complaints in left ventricular failure tending to be most severe in instances of severe failure Pressure on the bronchi or trachea by an extremely *large heart or aortic aneurysm* may produce coughing *Hemoptysis* may occur in severe left ventricular congestive heart failure In *acute* left ventricular heart failure with pulmonary *edema there is an extensive escape of erythrocytes into* the pulmonary edema fluid producing homogeneous [illegible] sputum [illegible] ly and is [illegible] Mitral [illegible] with hemoptysis An aneurysm may erode the bronchi or trachea and cause rather extensive hemorrhage with expectoration of pure blood in small or large quantities

7 **Miscellaneous Symptoms** — There are many symptoms which are related to cardiac dysfunction *Vertigo insomnia headache irritability hoarseness giddiness and faintness* are among the numerous symptoms experienced by these patients

THE PHYSICAL EXAMINATION

The examination of a *patient* with cardiac disease should be as thorough as that for any other patient The student should acquaint himself thoroughly with physical diagnosis no attempt will be made here to review completely the problem of physical examination of a patient A standard monograph on physical diagnosis may be consulted for a more

complete survey of the problem *The entire physical examination is important, and not one phase can be eliminated* In the examination of the cardiovascular system, certain phases of the study are of particular importance in interpretation of the state of the heart

1 **General Appearance** —The general appearance of the patient, such as his age, sex, nutritional state, skin, pallor, cyanosis, and the like, is observed The physiologic and chronologic ages of the patient are estimated Then certain specific types of examinations are made

2 **Pulse** —The pulsations of the main arteries available for examination are studied, including the radial, brachial, axillary, femoral, popliteal, dorsalis pedis, posterior tibial carotid and temporal arteries They are examined in the same fashion in which the radial artery is usually studied at the bedside The tips of the four fingers of the examining hand rest upon the radial artery with the index finger distal, while the rest of the hand grasps the patient's wrist The artery is felt for thickness, tortuosity, compressibility and fullness of the pulse The circulation through the artery is obstructed by applying firm pressure with the tip of the little finger (fourth) and the radial artery, distally, is explored with the tips of the middle and index fingers Normally this distal portion is not, or is just barely, perceptible With thickening of its wall, the artery remains palpable distal to the point of compression With severe arteriosclerosis, it feels like a rigid tube (*pipe stem*) The student should examine the radial and other arteries of many normal people of all age groups in order to become thoroughly acquainted with the normal variations with age

The rate and fullness of the pulse are noted Various types of pulses, depending upon the variations in the volume and timing, are shown in figure 46 Examination for pulsations in the abdominal aorta, iliac femoral, and popliteal arteries aids in the diagnosis of coarctation of the aorta or thromboembolic phenomena involving these vessels The former is unlikely to be present or, at least to be of clinical significance, if these arterial vessels pulsate well and it is surely simpler to palpate routinely for them in all patients with arterial hypertension if routine recording of arterial pressure in the legs is not possible

3 **Capillary Pulsations** —The capillary pulsations are then observed This may be done by several methods

(*a*) *Pressing down on the edge of the finger or toe nail* to produce a small area of blanching of the subungual area With pulsations of the capillaries, this blanched area decreases in size with each systolic phase of the pulse and increases during the diastolic phase Normally this is difficult to observe

(*b*) *Transillumination* of the finger tips with a small pocket flashlight will usually reveal some capillary pulsations the finger tip darkening with each systole

(*c*) *Following pressure* on the forehead with a *glass slide* just sufficient to obliterate the skin vessels, there will be a return of skin color with each systole

(*d*) *Vigorous rubbing of the forehead* to produce redness will often show a varying degree of the erythema with each phase of the pulse











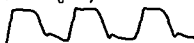






FIG 46 Diagrammatic representation of various types of pulse waves

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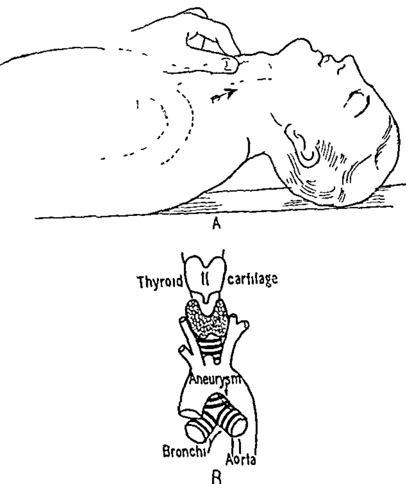


FIG 47 —Part A shows the method of eliciting a tracheal tug (*Oliver's sign*) Part *B* shows the manner in which dilatation of the aortic arch particularly the inferior surface pulsates against the left bronchus This is particularly likely to occur when there is a saccular aneurysm of the inferior portion of the arch of the aorta

CYANOSIS

Cyanosis is an important manifestation of cardiac disease It is due to discoloration of skin caused by the presence of reduced hemoglobin in the cutaneous vessels When there is cyanosis, at least 5 grams (6 to 7 volumes per cent) of reduced hemoglobin is present per 100 cc of blood In heart disease it is usually due to the following mechanisms

(*a*) **Impairment of Oxygen Exchange in the Lungs** —This is usually brought about by the edema fluid of left ventricular failure which covers the respiratory epithelium with a thin film of fluid interfering with the

4 **Ophthalmoscopic Examination** —An ophthalmoscopic examination should be done routinely A great deal of pertinent information about the cardiovascular system can be obtained from this examination Some of the earliest quantitative data in arteriosclerosis hypertension renal disease and the like can be obtained The student should begin to study eyegrounds in all of his patients as early as possible in his clinical career This should be done in a dark room and with the patient's pupils properly dilated (One drop of 2 per cent aqueous solution of homatropine should be placed in each eye every fifteen minutes until 3 drops have been used) Caution should be employed to avoid producing glaucoma from this drug

5 **Neck** —The neck is examined for thyroid enlargement arterial and venous abnormalities and tracheal tug (Oliver's sign) In testing for a tracheal tug the patient's head should be dorsiflexed slightly in order to make the larynx more prominent and to pull the trachea upward The inferior end of the anterior surface of the larynx is pushed and held in a cephalad position by the tips of the thumb and index fingers in order to stretch it *slightly and to pull the left bronchus closer to the inferior surface of the* aortic arch (Fig 47) If there is a tracheal tug there will be a tugging downward of the larynx with each systolic ejection of the heart This sign is encountered in aneurysms of the aortic arch

Kinking of the Carotid Arteries —This occurs frequently in arteriosclerosis or syphilis of the aorta with dilatation and uncoiling of the aorta and displacement upward of the roots of the arteries originating from the arch of the aorta In arteriosclerosis of the carotid arteries also there is bending and folding of the vessels to a greater extent (Fig 48) The kinking of these arteries results in turbulence of flow with resultant systolic and diastolic murmurs over the vessels Systolic and diastolic thrills may be associated with the murmurs With kinking and bowing anteriorly they can be seen to pulsate on inspection and can readily be felt on palpation This usually occurs in the right common carotid artery and is most often observed in senile arteriosclerosis *These changes are often confused with syphilitic aneurysm*

6 **Thorax** —The thorax should be examined carefully for changes related to the heart Deformities of the chest such as kyphosis *old fractures and* operative deformities tugging of the intercostal spaces barrel shaped chest of emphysema and the like should be noted as the heart may be *displaced* greatly with resultant impairment of its function The veins of the thorax and shoulders are often distended in disease of the heart and its great vessels Pressure from an aortic aneurysm may obstruct venous return and result in an increase in venous pressure with distention of the superficial vessels of the thorax

7 **Abdomen** —The abdomen is examined for ascites hepatomegaly splenomegaly distended veins a pulsating liver aneurysm of the abdominal aorta and other *important* changes

8 **Extremities** —The extremities are examined for vascular disease pallor cyanosis edema arthritis nodules cutaneous lesions and the like

to cause cyanosis

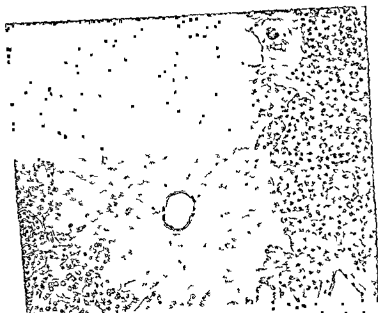


FIG 49 — Drawing of degenerative changes in the central portion of a liver lobule produced by venous congestion in congestive heart failure

JAUNDICE

Jaundice to a small degree at least, occurs frequently in heart disease with right ventricular congestive heart failure With this type of failure the liver is congested The stasis interferes sufficiently with
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transfer of O_2 from the alveolar air into the blood. Tracheal or bronchial obstruction such as is encountered in aneurysms may interfere with the flow of O_2 to the alveolar spaces. Associated diseases of the lungs such as chronic emphysema, bronchiectasis and chronic tuberculosis contribute to that already *produced by associated right and left ventricular failure of* the heart. In the usual patient hyperpnea associated with the dyspnea results in more than adequate oxygenation of the blood circulating through the lungs.

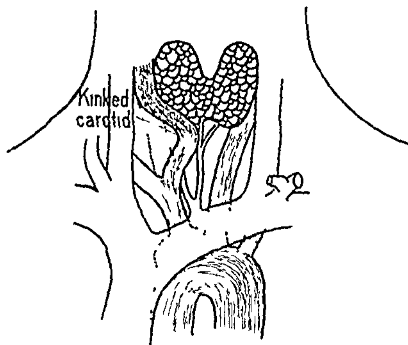


FIG. 18.—Illustration of kinking of common carotid artery produced by dilatation of the aorta at the origin of the innominate artery. This pushes the innominate artery cephalad. The common carotid, since it is relatively long and free of branches for a great distance, kinks. For obvious anatomic reasons, it almost always bends medially near the level of the suprasternal notch.

(*b*) **Stasis in the Peripheral Blood Vessels.** In heart failure, stasis of blood occurs in the peripheral blood vessels. The delay and sluggish circulation of the blood allows the tissues to extract more than the usual amount of O_2 from the hemoglobin. The residual reduced hemoglobin is increased in concentration to a level sufficient to produce a variable degree of cyanosis. The same phenomenon may occur with venous stasis from venous obstruction, such as occurs with extreme cardiac enlargement or mediastinal aneurysm. *Peripheral circulatory collapse*, as occurs with the shock encountered in congestive heart failure or myocardial infarction, will also produce stasis in the capillaries, venules and small veins, resulting in cyanosis.

tion of the ribs with cardiac systole, and bulging of the precordium *Thrills* are located by palpation For this purpose any portion of the palmar surface of the hands and fingers may be used, the hypothenar region often being the most sensitive Movement of the thoracic wall or limitation of movement may be noted by properly grasping the lower portion of the thorax bilaterally with both hands and noting the differential expansion

Thrills are often difficult to palpate, especially in obese patients or in patients with large breasts Considerable practice is required to detect faint thrills The student should not become discouraged by his initial failures to identify thrills for with perseverance and training, the necessary ability can be attained

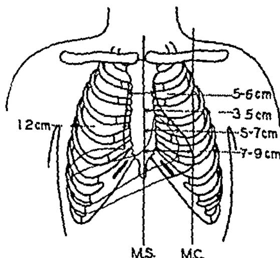


FIG 31 Location of the borders of the normal heart in relation to the midsternal line The distances in centimeters indicated are extreme values

Thrills that are faint ...
exercise to increase the ...
taken not to confuse a ...
produced by vigorous cardiac action The latter ... the thoracic wall

Thrills are timed similarly to murmurs Th... diastolic ... ommon ... a thrill is most often ... They

longed chronic right ventricular congestive heart failure This further disturbs excretion of bilirubin, causing retention and jaundice.

EXAMINATION OF THE HEART

Examination of the heart itself will be discussed somewhat briefly, with elaboration on certain pertinent phases which are often neglected

Inspection —The heart is inspected to locate the position of the *apex beat* This is normally in the fifth intercostal space medial to the midclavicular *line* *With enlargement, it is displaced to the left, or to the left* and downward In the obese patient or patient with large breasts, it is

[illegible] re easily [illegible] observer [illegible] P M I) may be similarly located Its normal location is shown in figure 50 These points of pulsation are displaced by changes in position, tending to be more medial in location when the subject is standing or sitting than when lying down

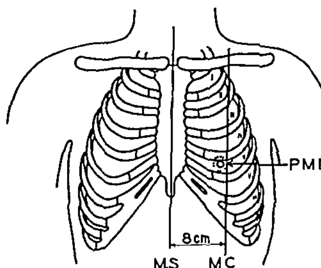


Fig 50 —Location of the point of maximal impulse (P M I) in the normal person

Retraction of the intercostal spaces in cardiac systole by adhesions (*Broadbent's sign*) may be observed Disturbances in movement of the diaphragm noted by *Litten's sign (peeling of the diaphragm from the* thoracic wall on inspiration) are of importance in cardiac disease

The precordial region of the thoracic wall bulges outward in extreme cardiac *enlargement* *An aneurysm located in the mediastinum* may also bulge outwardly and may be seen to pulsate or distend the local regions of the thorax with each systolic ejection of blood

Palpation —*Palpation* is employed in part to confirm many of the observations made on inspection, for example, the apex beat, P M I , retrac-

These sounds vary in character and intensity from area to area

(2) *Time* —The first and second sounds are separated from each other by a relatively short time interval in the average resting cardiac rate The second sound follows the first by a definitely shorter interval of time than the first follows the second (Fig 52) at normal and relatively slow rates When in doubt the *first sound* can usually be identified from the apex beat or the carotid pulse they all occur simultaneously If it is possible to identify the sounds in only one area the chest pieces of two stethoscopes may be used in such a way that one reaches each ear and with the known area as a reference the others may be explored and identified By noting the temporal relation of the sounds one may identify heart sounds in all areas



Fig 52 Phonocardiogram and diagram of the first and second heart sounds showing the relative temporal relationships

Phases of the Cardiac Cycle From the clinical point of view there are only two phases of the cardiac cycle

1 Systole
2 Diastole

These two phases are then identified from the heart sounds (Fig 53)

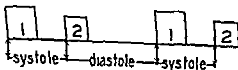


Fig 53 D gr of the first and s n l so

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Percussion —Percussion of the heart has been discussed previously There is a tendency among physicians to develop a defeatist attitude about percussion the advent of roentgenography has been mainly responsible for this attitude Certainly roentgenographic methods should always be employed whenever possible there is often no substitute for them Nevertheless no physician should fail to develop satisfactory ability to percuss accurately In many circumstances roentgenographic methods are not available particularly in acutely ill patients at home and the attending physician must rely upon percussion to detect cardiac size An immediate impression is often necessary and this can be furnished to a satisfactory extent by percussion Every student should practice and verify by roentgenographic means his ability to percuss accurately and *should never cease trying until ability has been adequately developed*

Percussion serves as a check of cardiac size previously determined by inspection and by palpation Not only should the apex be located but the cardiac border should also be completely outlined (Fig 31) Careful percussion will reveal distortions in cardiac configuration such as occur in mitral stenosis with enlargement of the left atrium and pulmonary conus upward and to the left It is difficult however to percuss the outline of the great vessels of the heart A good rule to remember is that the substernal *region above the heart is not really dull to percussion although it is not as* resonant as the pulmonary field Impairment of this resonance is accentuated (a definite dulness or flatness on percussion) in the presence of a *mediastinal mass*

AUSCULTATION OF THE HEART

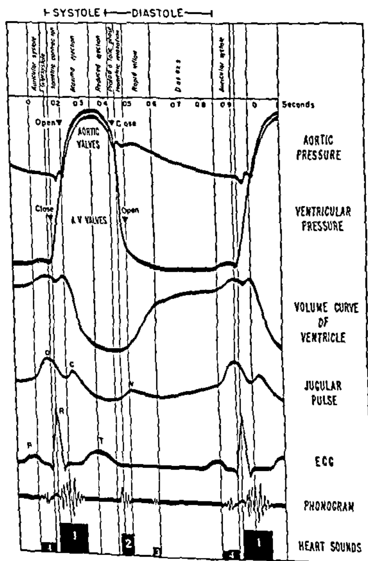
No phase of the cardiac examination is as important and yet is little understood as auscultation Many students complete their formal training and enter practice without ever achieving the ability to auscultate properly Many cannot identify or even time murmurs with sufficient confidence to remain in a branch of medicine in which the proper use of the stethoscope is exceedingly important Auscultation is simple and reliable but only if the physician learns the fundamental principles concerned with sound particularly those sounds encountered in the patient To learn auscultation by rote memory or cook book fashion spells defeat Practice in normal and abnormal persons is essential but only after the theoretic principles have been learned

The discussion of auscultation will be limited entirely to the heart and blood vessels Auscultation of the lungs is a separate and equally important aspect of physical diagnosis and *for that matter of cardiovascular diagnosis*

Heart Sounds The first problem that confronts the examiner when he *listens to the heart* is the identity of the heart sounds This is usually but not always simple As a rule the separate sounds can be identified in the average patient from their *quality* and the *timing*

(1) *Quality* —The characteristic *lub dub* of the first and second sounds is heard The first sound is usually more intense than the second at the apex

is complete and is normal. The student should train himself to listen only to systole and ign[illegible]
m[illegible] cular micro[illegible]
the microscope[illegible]



F[illegible] 5[illegible]
[illegible]mena. Th[illegible]
[illegible]ardiogram
[illegible] essential [illegible]
[illegible]

from the onset of the second sound to the time the first sound occurs is *diastolic* in time. Therefore to time anything in relation to the phases of the cardiac cycle, one must identify the heart sounds *first*. Murmurs, clicks, rubs, and so forth can thus be timed.

It is thus obvious that the phase of the cardiac cycle is determined by the phase of the cycle of the ventricles and not of the atria. Cardiac systole is *synonymous with ventricular systole, and the diastolic* phase of the heart is synonymous with the ventricular diastole. It is well to remember that there are also systole and diastole of the atria. The atria go into systole during ventricular diastole and into diastole during ventricular systole. Although such physiologic phenomena are well known, the student often forgets them in practice in the clinic and therefore cannot fail to confuse the hemodynamic *phenomena and is finally perplexed when considering* murmurs.

Third and Fourth Heart Sounds.—During the diastolic phase there may be two other sounds, the *third and fourth* (Fig. 54). The characteristics of these sounds, like those of the first, will be given under the discussion of sound tracings. The *third heart sound* occurs shortly after the second sound or early in diastole, and the *fourth heart sound* occurs late in diastole or just before the first sound, *i. e.*, just before systole begins.

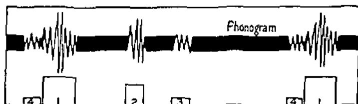


Fig. 54.—Phonogram and diagram of the four normal heart sounds showing their *relative intensities, pitch and time.*

The relationship of these sounds to other events in the cardiac cycle must be kept *in mind for their proper evaluation* (Fig. 55). This will be discussed in detail later.

Auscultation in Practice.—Auscultation of the heart must be performed in an organized fashion. It is better to begin at the *mitral area* first, identifying the *first* and *second* heart sounds. Once these have been identified, attention must be directed to the interval of time from the beginning of the first sound to the beginning of the second sound. The only sound usually heard *in the normal person during this period is the first sound*. This is followed by a period of silence, and then systole ends with the beginning of the second sound. Any sound heard during this period is systolic in time. This examination should be made during complete inflation and complete deflation of the lungs with the patient resting on his left side, leaning forward, standing, *sitting and lying supine, to be certain* that no other sounds or murmurs have been overlooked. If nothing other than the first sound is heard, then *the examination of systole for that area*

examine all of them with the patient at rest first, after exercise, and in various positions of the body. Obviously, it is necessary to coördinate the latter so that the patient does not exercise separately or change position ... the examination of each cardiac area separately ... with a ... d make ... nfusion ... with such murmurs

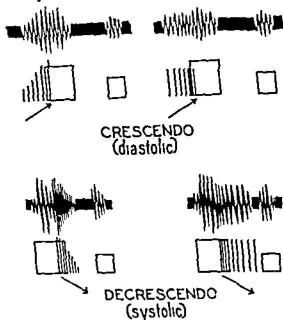


FIG 56 Phonogram and diagram of systolic and diastolic crescendo and decrescendo murmurs. A murmur may be *crescendo* (increasing) in *intensity or pitch* or it may be *decrescendo* (decreasing) in intensity and in pitch. Various combinations such as *crescendo-decrescendo* or *decrescendo-crescendo* murmurs may occur. The latter two combinations ... particularly likely to occur for the diastolic murmur of mitral stenosis.

Sound Tracings.—It is not our purpose in presenting this discussion of sound tracings to train the student in the interpretation of these records. They are used primarily to correlate he ... peripheral pulsations in such a way a ... the evaluation of a cardiac disorder ... sidered highly specialized or detailed but there is no other way to learn

should direct his attention to the period beginning with the second sound and ending with the beginning of the first sound and, as is true with the systolic evaluation, position and pulmonary expansion are important This is the diastolic period, which begins with the second sound If the period from the end of the second sound to the beginning of the first sound is silent, the diastolic evaluation is complete If any sound is heard during this usually silent period, it is diastolic in time A sound heard soon after the second sound should be studied to determine if it is a *third* heart sound, if it is heard late, a *fourth* sound should be considered A murmur heard during this period is diastolic Once each phase has been carefully examined separately, the two phases are coordinated With practice, this requires little time

TABLE 4 —TABLE OF TERMS TO BE USED IN DESCRIBING HEART SOUNDS AND MURMURS (From Nomenclature and Criteria for Diagnosis of Diseases of the Heart, New York Heart Association, Inc)

Intensity	*Pitch*	*Quality*	*Duration*	*Time*
		HEART SOUNDS		
Normal		Normal	Normal	
Faint		Sharp	Short	
Weak		Snapping		
Distant		Valvular		
Muffled				
Loud		Booming	Prolonged	
Accentuated		Muscular		
Increased				
Absent		Split		
Replaced by a murmur		Reduplicated		
		Ringing		
		Metallic		
		Bell-like		
		Tambour		
		Hollow		
		MURMURS		
Faint	High	Blowing	Short	Systolic
Soft				
Moderate	Medium	Harsh	Moderate	*Early Systolic*
		Rough		
		Coarse		
Loud	Low	Musical	Long	Late Systolic
		Rumbling		Diastolic
		Crescendo		Early Diastolic
		Decrescendo		Mid-diastolic
				Presystolic (late diastolic)

All murmurs are studied for their characteristics, *i e*, *intensity, pitch, quality, duration*, and *transmission* (Table 4 and Fig 56)

Once the mitral area has been adequately auscultated, then the *aortic, pulmonic* and *tricuspid* valvular areas in turn are examined similarly

Examine all of them with the patient at rest first, after exercise, and in various positions of the body. Obviously, it is necessary to coördinate the latter so that the patient does not exercise separately or change position for the examination of each cardiac area separately.

... the fourth heart sound is often confused with a ... therefore, should make ... to avoid any confusion with such murmurs.

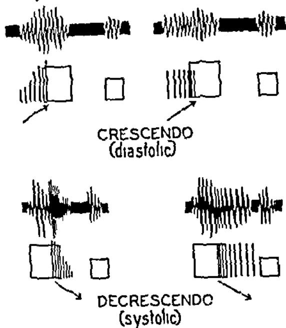


Fig. 56.—Phonogram and diagram of systolic and diastolic crescendo and decrescendo murmurs. A murmur may be *crescendo* (increasing) in *intensity or pitch* or it may be *decrescendo* (decreasing) in intensity and in pitch. Various combinations such as *crescendo-decrescendo* or *decrescendo-crescendo* murmurs may occur. The latter two combinations are particularly likely to occur for the diastolic murmur of mitral stenosis.

Sound Tracings.—It is not our purpose in presenting this discussion of sound tracings ... They a... periphe... the ev... considered unduly specialized or detailed but there is no other way to learn

the subject properly It is hoped that the student will *think* carefully, thoroughly and clearly as he reads the following discussion on sound tracings He will be rewarded for his efforts in practical clinical auscultation and, in addition can easily advance to an intelligent interpretation of sound tracings

ASPECTS OF PHYSICAL CHARACTERISTICS OF SOUND

Audible Frequencies —*The audible frequencies* of sound vary between 16 cycles per second and 20,000 cycles per second The *intensity*, measured in dynes per square centimeter per second, must be sufficient to be audible at any given frequency The duration of the sound is also important in audibility For example, at frequencies of 32 to 2,500 cycles per second *two cycles* are sufficient for perception Sounds with frequencies below those audible by man are called *infrasonic*, and those above audible levels are known as *ultrasonic* or *supersonic*

Auditory sensations are classified as

1 **Tone** — A tone is a sound wave which is sinusoidal in nature (Fig 57) and may be expressed by the formula

$P = P_0 \sin \omega t$

where P = value at time t

P_0 = initial pressure in dynes per square centimeter per second

$\frac{\omega}{2\pi}$ = frequency

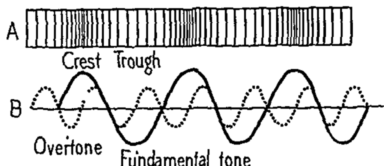


Fig 57 —Diagram showing how a sound wave is propagated with a trough and crest The fundamental tone and an overtone are shown in Part *B*

2 **Sound** — A sound consists of a *fundamental tone* with a number of superimposed *overtones* The frequencies of the overtones are multiples of the fundamental tone (Fig 57) Such a combination has the formula

$$P = P_1 \sin (\omega t + \phi) P_2 \sin (2\omega t + 2\phi) -$$

When there are various sounds existing simultaneously whose fundamental frequencies are not multiples of each other, a *mixed* sound is said to exist This may be expressed by

$$P = \sum_{1}^{\mu} P_n \sin(n\omega_1 t + n\phi) + \sum_{1}^{r} P_r \sin(m\omega_2 t + m\phi) -$$

Noise.—A noise is a sound consisting of irregular vibrations. From the foregoing brief discussion it is obvious that the terms "heart tones" and "heart sounds" are misnomers. The French clinicians are correct in referring to them as "bruits" (*heart noises*). Nevertheless to conform with usage the term *heart sounds* will be employed throughout these discussions in spite of the technical indefiniteness of the term.

Characteristics of Heart Sounds.—Heart sounds have the following characteristics (Fig. 58):

1. *Intensity* = force or amplitude of the vibrations.
2. *Pitch* = frequency of the vibrations per unit time.
3. *Duration* = length of time it persists.
4. *Timbre* = Quality which depends upon overtones or harmonics accompanying the fundamental tone or note. This characteristic is present only if the heart sound should exist as a sound in the true physical sense.

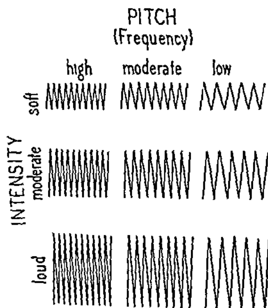


FIG. 58.—Illustration showing how a sound may vary in intensity and pitch. A sound for example may be faint or of low intensity but high in pitch, or loud and low in pitch, etc. The various combinations and their significance must be known to appreciate the importance of heart sounds in clinical cardiology.

Origin of Heart Sounds.—Heart sounds originate from:

1. **Heart Muscle.**—When a muscle contracts, sound waves are initiated by the contracting fibers (Fig. 59). This is true of cardiac muscle as well as skeletal muscle.

2. **Heart Valves.**—When the heart valves *open* as blood flows through them or when they *close*, and especially with sudden snapping of the chordæ tendineæ, sound waves are set into motion (Fig. 59).

3. **Flowing Blood.**—*Blood which is flowing rapidly* through chambers of irregular size, such as the heart chambers and great vessels, produces *turbulence* of flow with resultant initiation of sound vibrations.

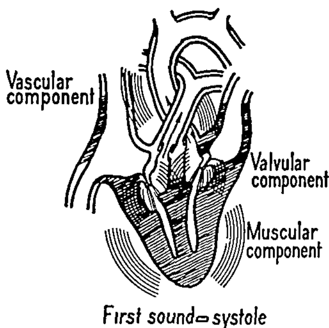


Fig. 59.—The origin of the vibrations which produce the first heart sound. (See text.)

4. **Vascular Walls.**—Sudden variations in intra-arterial tension set the vascular walls into vibration, resulting in production of sound (Fig. 59). Turbulent flow will also cause the vascular walls to vibrate.

The transmission of heart sounds varies with the characteristics of the surrounding tissue, especially its *natural frequency*. If this is of the same order as the frequency of the heart sounds, it will be intensified by *resonance*. If the *natural frequency* differs greatly from that of the heart sound, it will be weakened by *interference*.

It is obvious that the *intensity* of the heart sounds depends upon: (1) *the position of the heart*, (2) *the nature of the surrounding structures*, and (3) *the position of the stethoscope* in relation to the source of the sound.

The heart sounds reaching the ear or recorder differ from the original and therefore represent "modified sounds."

Extraneous Sounds.—During auscultation or the mechanical recording of sound many extraneous or extracardiac sounds may be encountered such as friction rubs, friction between chest piece and hair or skin, râles, skeletal muscle sounds, etc. The student must train himself to recognize these sounds and to ignore them as well as the many other existing in an examination room.

RECORDING OF SOUND

With the advent of more satisfactory and practical methods, the mechanical recording of sound has become more and more important within recent years. The chief value of such recordings, although still not fully satisfactory clinically, is concerned with the learning and understanding of normal and abnormal heart sounds. The actual identification of various types of murmurs can be attained only at the bedside and on normal people.

The Recording Devices.—Many types of recording devices are available: some are *direct* or mechanical with the sound wave being transmitted to a vibrating membrane, whereas others are *indirect*. The latter usually consist of suitable microphones with vacuum tube *amplification* and electrical

to be suitable must have
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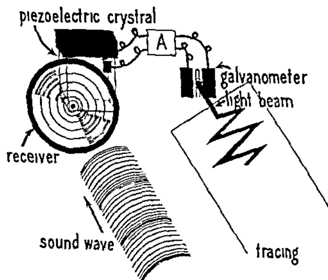


Fig 60 A
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20 and 150 cycles per second. These recorders must have a minimum of inertia. The essential requirements of mechanical sound recorders are:

1. High sensitivity with low threshold of intensity *and frequency*.
2. Deflections that are *proportional* to the acting forces.
3. Low inertia.
4. Adequate damping.
5. A natural frequency at least *five times that of the most rapid* frequency to be recorded.

The frequency threshold varies from 20 to 40 cycles per second depending on the type of recorder.

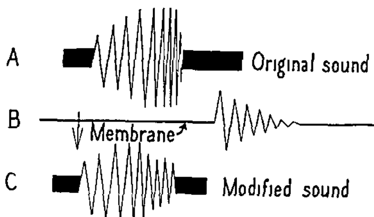


FIG. 61. Any matter or object when suddenly set into vibration will vibrate with a frequency determined by the conditions existing at the time. Regardless of how hard or lightly the object is struck, it always vibrates with the same frequency (*natural frequency*) provided the same conditions are maintained. If such an object or matter is to transmit sound without distortion, it must have a natural frequency which is at least five and preferably six times the highest frequency of vibration to be transmitted. Part A shows an original sound with an increasing pitch. The pitch reaches a level greater than that of the natural frequency of a membrane, B, intended to record properly the original sound. Part C shows that the final record is tracing with the high pitched portion of original sound improperly recorded as to frequency or pitch and intensity. Such distortions occur when inferior recording devices are employed. Furthermore, the tissues *of the chest constantly distort the original sound produced at the site of formation* so that the sound that finally reaches the surface of the thoracic wall is considerably different from the original sound.

THE PHLEBOGRAM

The phlebogram is still an important record in cardiology. It serves a definite purpose in the understanding of hemodynamics and cardiodynamics and certainly the origin of heart sounds. The relation of the phlebogram to the physiologic phases of the cardiac cycle is shown in figure 62. This should be thoroughly mastered by the student.

The positive *a* wave is due to systole of the right atrium which causes some blood to regurgitate into the jugular vein. The positive *c* wave is produced by right ventricular contraction which causes the tricuspid valve

to bulge into the right atrium and in turn displaces blood into the jugular vein. The positive v wave is produced by filling of the right atrium which is closed below with overfilling into the jugular vein.

PHYSIOLOGIC PHASES OF THE CARDIAC CYCLE

The physiologic phases of the cardiac cycle should be known in order better to understand heart sounds and their relation to cardiodynamics.

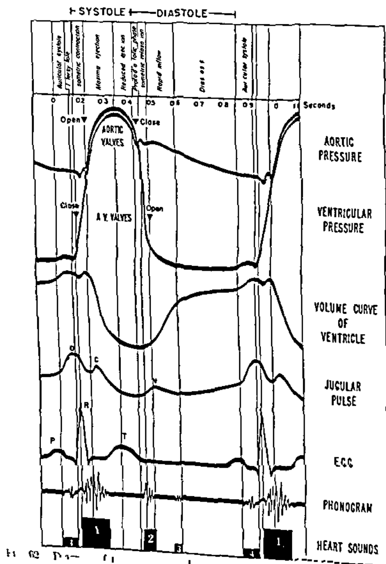


Fig. 62

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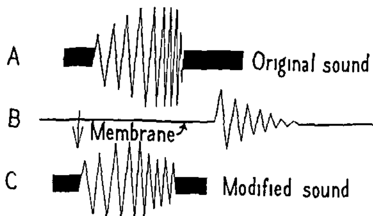


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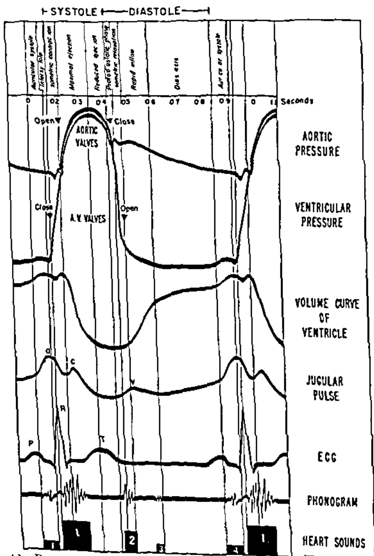


Fig. 42. Diagram of the relation of the components of the phlebogram to the heart sounds. This should be learned and understood.

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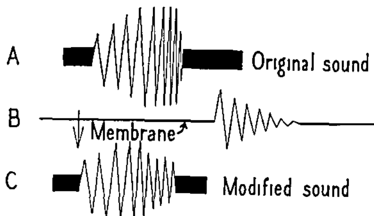


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2 Variations of the sounds from cycle to cycle
3 Characteristics of each sound as to
   (a) Duration
   (b) Amplitude of vibrations
   (c) Frequency of vibration
   (d) Variations with respiration
   (e) General characteristics
4 Variations with areas explored
5 Principal temporal relations to events in the reference tracings (electrocardiogram and phlebogram)

Ordinary clinical auscultation does not permit such a complete analysis The deficiencies are obvious

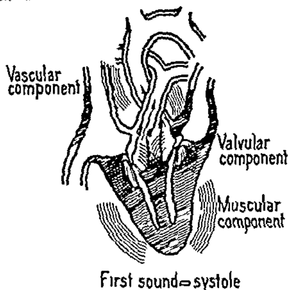


Fig 63 Diagram showing the components of the first heart sound

NORMAL HEART SOUNDS

First Heart Sound The first sound is produced by the following phenomena listed in descending order of importance

1 Muscular contraction and sudden tension of the ventricular wall (*muscular factor*) (Fig 63) with shortening produce sound just as does contraction of a skeletal muscle This can be verified by listening with a stethoscope over the biceps muscle as it contracts The friction between the contracting fibers may be responsible for some of the muscle sounds
2 Sudden closure of the atrioventricular valves (*valvular factor*) results in sound (Fig 63) This is said to be due to valves and chordae tendineae snapping taut

textbooks of physiology should be consulted for further details. These phases are (Fig. 62):

1. *Presystole* or *auricular systole* begins with the contraction of the atria. This normally lasts 0.10 second.
2. *Intersystole* begins with the end of the contraction of the atria and ends with the beginning of ventricular contraction. It lasts about 0.04 second.
3. *Ventricular systole* has *two* distinct phases:
   (a) *The phase of isometric contraction*, or the period when the muscle is gathering force and increasing its tone but before the fibers have shortened; and
   (b) *The phase of ejection*, with *three subdivisions* which are self-explanatory:
      (1) Minimal
      (2) Maximal
      (3) Reduced
4. *Ventricular diastole* has *four* phases:
   (a) *The protodiastolic phase* lasts about 0.04 second and exists between the moment of the beginning of ventricular relaxation and the closure of the semilunar valves.
   (b) *The isometric relaxation phase*, or the moment during which the ventricular muscle fibers are relaxing in force but not in length. Blood is not entering or leaving the ventricles, and the semilunar valves are closed.
   (c) *The rapid filling phase* lasts 0.11 second. At this moment the atrioventricular valves are open and blood is entering the ventricles from the atria.
   (d) *The slow filling or diastolic phase* represents the period between rapid filling and the beginning of the next auricular contraction.

The student should not only become acquainted with these phases but should know *direction, rate and force of the blood moving in the heart* at these various times. He should also know the state of the valves and cardiac muscle during each phase and during the transition from each phase. Such knowledge is essential for an understanding of heart sounds and murmurs. Sound tracings are usually recorded with the phlebogram and electrocardiogram as references to make possible necessary correlations with other cardiac events. These *correlations* require thought and knowledge of cardiac physiology. The study of sound tracings does not actually differ a great deal from the ordinary study of heart sounds and murmurs with the stethoscope. Since both are necessary and valuable, they will be presented in discussions to follow.

ANALYSIS OF THE PHONOCARDIOGRAM

The sound record is analyzed for:

1. Number of sounds per cardiac cycle.

occurs just after intersystole and precedes the onset of the *c* wave of the phlebogram (Fig 62)

The second component (Fig 65) is separated from the first by a short interval. It is identified by its *higher frequency and greater amplitude*. It *begins with or shortly after the peak of the usual R wave and ends before the onset of the c wave*, which represents the beginning of the ejection phase. It is probably due to the closure of the A-V valves (Fig 62). It has about 1-4 vibrations.

The third component (Fig 65) is almost always clearly separated from the second component. It begins with the beginning of the *c* wave of the *jugular pulse tracing (the ejection phase)*. It is associated with the *opening of the semilunar valves* and the *minimal* ejection phase. It usually has *two vibrations of high frequency and high amplitude*.

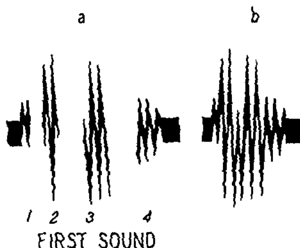


Fig 64 Part *a* shows the four components of the first heart sound separated, whereas part *b* shows them together as they actually occur. The *first* component occurs during the period of isometric contraction; the *second* during the period of closure of the A-V valves and beginning of the ejection phase; the *third* during the period of minimal ejection; and the *fourth* during the *maximal ejection phase* or period of acceleration of blood flow. Consult the text for details.

The fourth component (Fig 65) follows the third component without an intervening interval. It is low in frequency and low in amplitude and may be represented as a *blurred line*. It occurs simultaneously with *the peak of the c wave of the jugular pulse* and the *anacrotic notch of the arterial pulse* (Fig 62). *It seems to be related to the sudden acceleration of the blood flow in the main arterial vessels during the maximal ejection phase.*

The *first sound* has an average *frequency* of 50 vibrations per second, although observers have found it to vary from 25 to 35. Its *duration* varies from 0.10 to 0.17 second.

3 Movement of the blood and sudden distention of the arteries at the base of the heart (*vascular factor*) result in turbulence of flow and vibration of the vascular walls with production of sound (Fig. 63)
4 Residual vibration from auricular contraction (*auricular factor*) often contributes to the first sound whenever the auricular contraction or fourth heart sound occurs late in the cardiac cycle (Fig 64)
5 Some observers contend, although it has not been proved, that Korotkow sounds are produced in the semi-occluded coronary vessels which are being squeezed by the tightening ventricular muscle

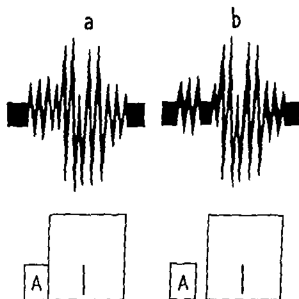


FIG. 64 —Part *a* shows the auricular or fourth heart sound merging with the first heart sound. This often results in an error of interpretation by the clinician. The

The graphic appearance of the first sound (Fig 64) is not a configuration that the observer would have predicted by his experience with auscultation. This is due in a large part to the *traditional* practice of representing the sound by a solid rectangular column (Fig 64). Phonocardiography has aptly filled this gap and taught us a great deal. The first sound actually consists of four components.

The first component (Figs 64 and 65) consists of a single small, relatively low frequency, thick or slurred vibration (at times there are two) which *immediately follows the auricular sound* when the latter is present. It begins in all areas about 0.011 to 0.039 second after the *QRS* complex has begun and about 0.008 second before the peak of the usual type of *R* wave. This component occurs as a result of the *isometric contraction* and therefore

2 The first sound *precedes the apex beat* by 0.01 second when it is recorded at the apex of the heart and by 0.05 second when recorded at the base of the heart in the second intercostal space

3 *All vibrations preceding the QRS complex are nonventricular in origin*

4 The arterial pulse waves and heart sounds show poor correlation

5 The first sound obviously precedes the rise in the fontanellar pulse

Second Heart Sound — The *second sound* (Fig. 66) is *shorter and higher pitched* than the first sound which is relatively *prolonged and low pitched*

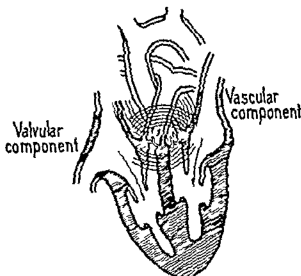


Fig. 66 The origin of the vibrations responsible for the second heart sound

Graphic characteristics of the second sound The principal and high amplitude vibrations are preceded by a slow oscillation which appears before the incisura or dicrotic notch of the carotid arterial pulse and occurs within the *protodiastolic phase* of the cardiac cycle (Fig. 66) There is really *one component* which has *four to five* vibrations with a frequency of 34 to 41 per second It is produced mainly by *closure of the semilunar valves* and vibrations of them and the root of the aorta and pulmonary artery

Relation of the Second Sound to Other Cardiac Phenomena 1 The second sound *corresponds with the first auscultatory or clinical evidence of a decrease in intraventricular pressure* It occurs with closure of the semilunar valves (Fig. 67)

2 It usually begins 0.01 to 0.05 second *after the end of the T wave* of the *electrocardiogram* It may precede the T wave by [illegible] second because of the great tendency of the T *wave to vary* in shape and duration within the cardiac cycle (Fig. 66)

Relation of the First Sound to Other Cardiac Events — 1 The beginning of the first sound and the increase in intraventricular pressure coincide closely (Fig 66) If it precedes the beginning of the *isometric contraction phase*, this is explained by the presence of the auricular component or residual of the fourth heart sound

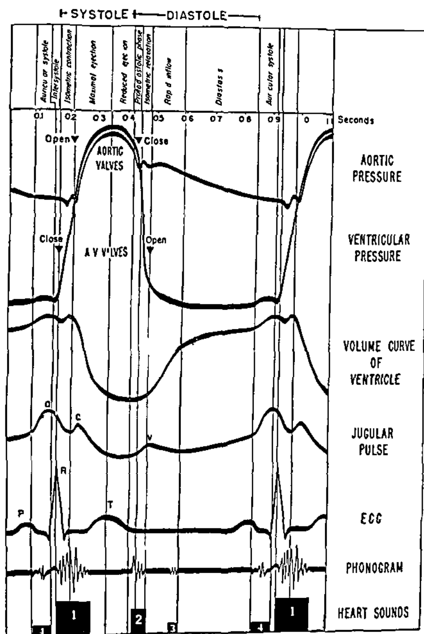


Fig 66 —Diagram of the relationships of the second heart sound to the other sounds and cardiac phenomena The student must understand these physiologic facts to understand properly cardiac function

The Fourth or Auricular Sound.—The graphic records (Fig. 69) show that the auricular sound is frequently present in the normal heart but this sound is not always heard on auscultation. It is easily missed because the auricular (fourth heart) sound is of: (1) *low intensity*, (2) *short duration*, and (3) *low frequency*.

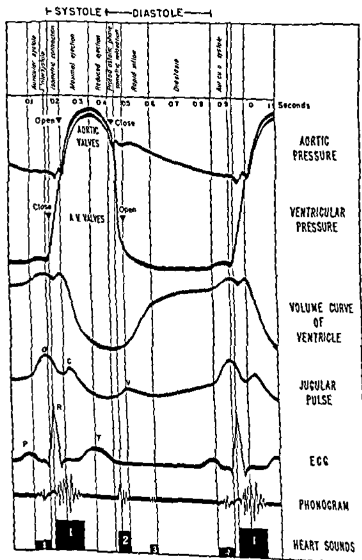


Fig. 68. The third heart sound and its relationships to other cardiac hemodynamic phenomena.

3 It begins with the lowest part of the *dicrotic notch* of the *carotid pulse*

4 The beginning of the second sound *precedes* the summit of the *v* wave by a *constant* interval of about 0 11 second

5 The second sound is *split* in about 90 per cent of normal people due to asynchronous closure of the aortic and pulmonic valves The *first* component is aortic valvular in origin and the second component is of pulmonic valvular origin

Third Heart Sound — *The third heart sound* is probably produced by the inrush of blood into the ventricles from the atria producing vibrations in the walls of the ventricles *It is frequently confused with a reduplication of the second sound* This sound is present in about 85 per cent of individuals from ten to twenty years of age *It decreases with age and is not often* heard in young children

Graphic characteristics of the third sound (Fig 68) The third heart sound occurs late in the period of rapid ventricular filling It is seen just *before the final portions of the descending limb of the v wave of the jugular pulse tracing* The interval between the second and third sounds represents the period of time between closure of the semilunar valves and the final stages of rapid ventricular filling This time is fairly constant ranging from 0 11 to 0 14 second The *amplitude* of the *third sound* is usually but *not always* less than that of the second sound Its *duration* averages 0 08 second (range 0 07 to 0 10) The *frequency* varies between 25 to 35 vibrations per second The number of vibrations varies from three to five

Relation of the Third Sound to Other Cardiac Functions 1 The third sound occurs after closure of the semilunar valves

2 It occurs near the end of the *v* wave of the venogram

The character of the third sound depends upon several factors most of which are particularly active during congestive heart failure with a dilated "flabby" heart and venous and atrial hypertension The character of the third heart sound depends upon

1 *Suddenness of ventricular filling*
2 Condition of the ventricular musculature and its ability to respond to the inflow of blood and
3 *Degree of contact of the heart with the thoracic wall*

The suddenness of ventricular filling depends upon the difference in pressure in the atria and ventricles and the emptiness of the ventricles It is obvious therefore why *abdominal pressure elevation of the legs increase in heart rate* and so on *facilitate perception of the third heart sound* It is also easy to see why abnormal physiologic conditions such as *congestive heart failure* result in a loud third heart sound and *protodiastolic gallop rhythm* a common observation in congestive heart failure In congestive heart failure the venous pressure is high therefore the blood rushes into the ventricles which are flabby causing their walls to vibrate easily The dilated ventricles are close to the thoracic wall and the sounds produced are thus readily transmitted to the stethoscope

4 Distention and vibration of the ventricular walls by the blood entering from the atria
5 Friction of the atria against the surrounding tissues

Graphic characteristics of the fourth heart sound

1 The vibrations begin with the onset of auricular contraction starting 0.04 to 0.06 second after the peak of the *P* wave This

2 tention of the

3 *P* wave

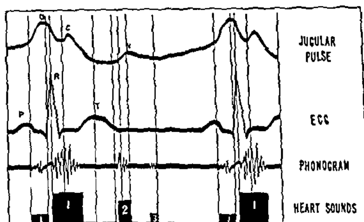


Fig 70 The fourth heart sound and its relationship to the venogram and electrocardiogram

The *fourth heart sound* is found on graphic records of about 93 per cent of normal medical students 36 *per cent of infants* 69 *per cent of school*

into this sound The fourth heart sound has *a frequency of about 20* cycles per second It thus has the *lowest pitch* of all the normal heart sounds It is best recorded in the mesocardial *area but* is loudest when recorded

it begins 0.018 to 0.064 second (mean 0.043±0.0054) *after* the summit of the *P* wave in lead II In infants it begins 0.06 second after the peak of *P*

temporal relations since the *P* wave is so variable from person to person and from time to time

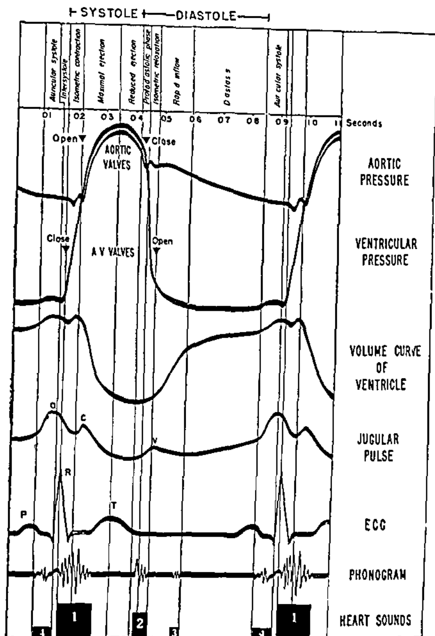


Fig. 69 — The fourth heart sound and its relationships to other cardiac hemodynamic phenomena

The *auricular sound is produced* by

1. Auricular contraction and shortening of the muscle fibers
2. Tensing and vibration of the auricular wall
3. Flow of blood through the atrioventricular orifices with the associated turbulence

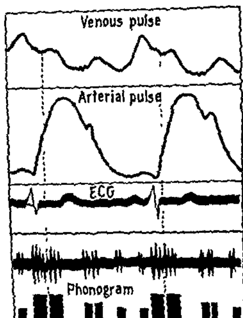


Fig. 73.—Splitting of the first heart sound due to wide separation of the second and third components of the sound. The relation to other cardiac phenomena is shown. Consult text for details.

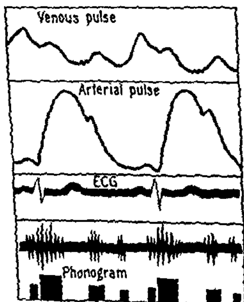


Fig. 74.—Splitting of the first sound due to a loud fourth or auricular sound. Note the relationships to other cardiac phenomena.

SPECIAL TYPES OF VARIATIONS IN THE HEART SOUNDS

Certain types of variations in heart sounds which are of practical significance in clinical medicine will be discussed. This is not intended to be complete and does not include many of the rare varieties that may be encountered. Such abnormalities are problems for more advanced study.



Fig. 71 — Physiologic splitting of the first heart sound due to accentuation of the second and third components. This apparently is the only type of splitting that occurs in the normal state.

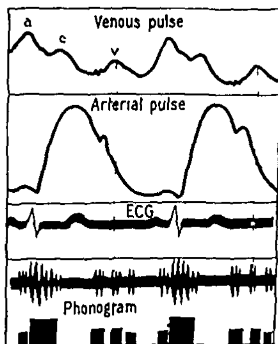


Fig. 72 — Splitting of the second heart sound due to the "opening snap" of the mitral valve during early filling of the ventricle in diastole. The relationship of the various heart sounds to other hemodynamic and electrocardiographic phenomena is shown.

asynchronous closure of valves. Many are instead due to separation of the two main components of the first ... influences (Figs. 71 and ... in the normal person

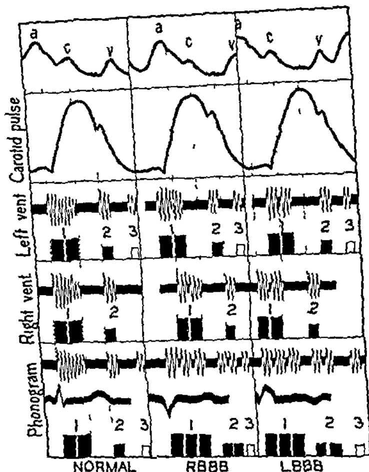


Fig. 76. Th... block. Remem... left ... left

At times an accentuated auricular or fourth heart sound is erroneously considered as splitting of the first sound (Fig. 74). Another excellent example of splitting is found in *bundle branch block*. In bundle branch block one ventricle contracts earlier than the other. As a result of this there is an asynchronous production of the components of the heart sounds. The changes brought about are shown in figure 75. This figure requires

Splitting of Heart Sounds.—It was not until the advent of phonocardiographic methods that the problem of splitting of heart sounds was *understood*. Many *concepts based on clinical auscultation alone* were later found to be erroneous. For example, the normal physiologic splitting of the first sound due to a loud auricular (fourth) sound and the pathologic

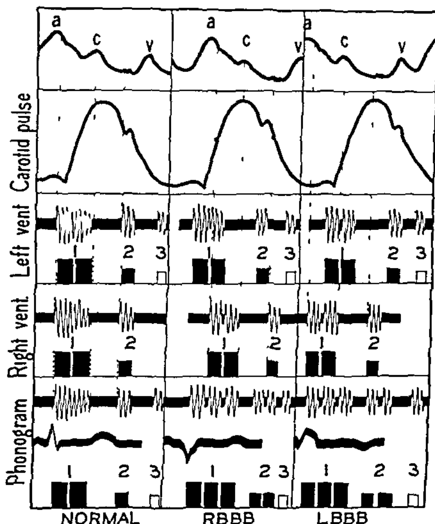


Fig. 75.—Splitting of the first and second heart sounds in right and left bundle branch block. Comparison is made with the heart sounds in the normal. This figure should be carefully studied and the hemodynamic disturbances should be properly evaluated and learned. Remember that the right side of the heart is responsible for the venogram and the left ventricle for the carotid pulse and most of the heart sound.

splitting *of the second* sound due to the opening snap of mitral stenosis (Fig. 72) are more clearly understood today with the use of graphic methods.

Splitting of the First Sound.—Since the development of cardiophonography, it has been found that many instances of splitting are not due to

asynchronous closure of
the two main component
influences (Figs 71 and
in the normal person Similar splitting occurs in various disease states

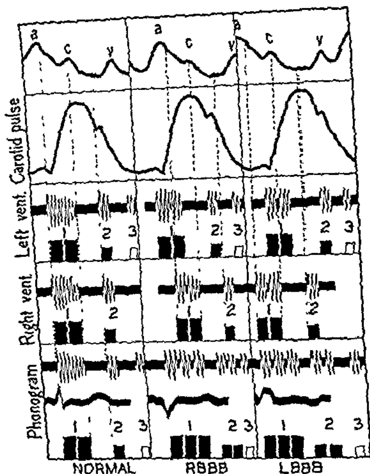


Fig 76 The displacement of the third heart *sound in right and left bundle* branch block Remember the right side of the heart is responsible for the venogram and the left ventricle for the *carotid pulse* and most of the heart sounds

At times an accentuated auricular or fourth heart sound is erroneously considered as splitting of the first sound (Fig 74) Another excellent example of splitting is found *in bundle branch block* In bundle branch block one ventricle contracts earlier than the other As a result of this there is an asynchronous production of the components of the heart sounds The changes brought about are shown in figure 77 This figure requires

careful study to separate the hemodynamic entities that produce the components of the heart sounds in the asynchronous cardiac activities. It can be seen how the two main components of the first sound and the second sound produced by each ventricle separately occur synchronously in the normal heart and asynchronously in the presence of bundle branch block.

Splitting of the Second Sound.—Many examples of splitting of the second sound identified by clinical auscultation have since proved to be due to the close grouping of the second and third heart sounds and therefore are not true splitting. True splitting is the result of asynchronous closure of the aortic and pulmonic valves. This is usually found in the presence of bundle branch block (Fig. 75) and premature beats.

Bundle Branch Block.—Bundle branch block is the best etiologic example of splitting of heart sounds. The mechanism is simple to understand once the heart sounds and their mechanisms are learned. In bundle branch block the ventricle supplied by the intact bundle contracts first. It therefore produces its components of the heart sounds first. If this ventricle contracts first by 0.04 to 0.06 second, *three well developed components* of the first sound and *two* of the second are grouped together. The first and second components and their grouping are shown in figure 75. All of these figures and their legends should be carefully studied and the splitting should be correlated with the physiologic activity within the heart.

In bundle branch block the *third heart sound* is displaced in relation to the *v* wave of the venous pulse (Fig. 76). *In left bundle branch block* the sound appears later than the end of the descending limb of the *v* wave which represents the rapid filling phase of the right ventricle. This relationship exists because the right ventricle contracts first. *Remember that the left ventricle is mainly responsible for the heart sounds and the right ventricle for the venous pulse.* *In right bundle branch block* (*left ventricle contracting first*) *the third heart sound* coincides more or less with the summit of the *v* wave.

RELATION OF HEART SOUNDS TO TYPE OF PATIENT

There are certain variations in the heart sound which occur with variations in age of the patient and with pregnancy.

1. Age. *Fetal* heart sounds have been recorded but not to any practical extent.

In *infants* the second sound is not as clear as it is in adults. The second sound in the pulmonic area is louder than that in the aortic area. This is the result of the continued influence of the fetal type of circulation with the relatively high pulmonary blood pressure. As the person grows older aortic pressure becomes higher and the pulmonary blood pressure relatively lower. This results in an increase in the aortic second sound over the pulmonic second sound. The *third heart sound* occurs in about 18 per cent of infants. The *fourth heart sound* is also heard at times.

The *first sound* has 3 to 5 vibrations which rise quickly to a maximal amplitude. It lasts about 0.064 to 0.172 second (usually between 0.10 and 0.14 second). It has a frequency of 30–50 cycles per second.

The *second sound* has 2 to 6 vibrations and reaches a maximal amplitude gradually. Its duration varies between 0.045 and 0.164 second (usually between 0.0 5 and 0.100). It has a frequency of 31 to 64 cycles per second, the pitch being similar to that of the first heart sound.

Children have heart sounds similar to those of adults. The *third sound* is present in 83 per cent of children.

There has been no systematic study of heart sounds in *senility*. However, the intensity is found to be reduced.

2. **Sex**.—There are no apparent sexual differences, although there have been relatively few studies in women.

3. **Pregnancy**.—The *first, second* and *third* heart sounds are present in about 22 per cent of pregnant women, and all *four heart* sounds are heard in 6 per cent.

HEART SOUNDS IN DISEASE

... e influence of ... he changes in ...

Gallop Rhythm

Gallop rhythm (triple rhythm) is a clinical auscultatory phenomenon in which the sounds are so grouped and of such intensity as to produce the effects of a horse galloping in the distance. The rate of the gallop varies with the cardiac rate. It usually represents serious myocardial disease.

The *extra sound* is called the gallop sound. This appears under three circumstances:

1. It may coincide with auricular systole, appearing before the a wave of the jugular pulse, thus being an exaggerated *auricular* or *fourth heart sound*.

2. It ... the ... gera ...

3. The third and fourth heart sounds may coincide ... rates ... of ... re ...

1. Nomenclature of Gallop Rhythm in Terms of the Time of the Gallop S...

... an exaggerated vascular component, the nature of which is vague and indefinite.

(b) *Protodiastolic gallop rhythm* is present when the gallop sound occurs early in the diastolic phase of the cardiac cycle. It is due to an exaggerated third heart sound and is common in congestive heart failure (Fig 78). For further discussion refer to the sections on the third heart sound and the signs of left ventricular failure.

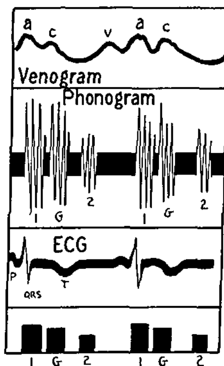


Fig 7 Systolic gallop rhythm showing the relationship of gallop sound to other cardiac phenomena. The mechanism of production of the gallop sound G is unknown.

(c) *Mesodiastolic gallop rhythm* is present when the gallop sound occurs in the middle of the diastolic phase of the cardiac cycle. It is found in fairly rapid cardiac rates and is due either to the *auricular or fourth heart sound* or to the *third heart sound* occurring in this phase of the cycle or to both (Fig 79).

(d) *Presystolic gallop rhythm* exists when the gallop sound occurs late in diastole or immediately preceding systole. It is due to an exaggerated *auricular or fourth heart sound* at normal or relatively slow rates (Fig 80).

2 **Nomenclature of Gallop Rhythm in Terms of the Mechanism Responsible for the Gallop Sound** —(Table 5.)

(a) *Presystolic or auricular gallop* constitutes about 27 per cent of all gallop rhythms.

(b) *Rapid filling gallop* (protodiastolic) constitutes about 14 per cent of all gallop rhythms.

(c) *Summation complete or incomplete gallop* (mesodiastolic) constitutes about 59 per cent of all gallop rhythms. This type of gallop is usually included by most physicians with protodiastolic gallop.

The term *protodiastolic* gallop rhythm should be reserved for use only in those patients with heart disease. The canter sound may be heard in normal people with a thin thoracic wall and a loud *third* heart sound.

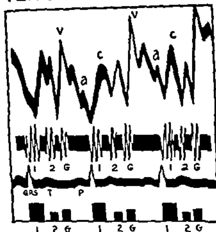


Fig. 78.—Protodiastolic gallop rhythm. The gallop sound G is really an accentuated third heart sound. Protodiastolic gallop rhythm is likely to occur in left ventricular congestive heart failure because the third heart sound is prone to be accentuated when the left ventricle fails. This is true because (1) the left ventricle is usually dilated and therefore close to the anterior thoracic wall, thereby transmitting the usually faint third

occur in toxic or degenerative states of the myocardium, such as are encountered in acute infections, severe anemias, or any type of organic heart disease associated with congestive heart failure.

HEART SOUNDS AND MURMURS IN VALVULAR DISEASE

It is impossible ...

... become acquainted with the other murmurs by a study of the medical literature, and he will be able to understand the mechanism of any murmur once he has thoroughly learned the fundamental mechanisms of their production.

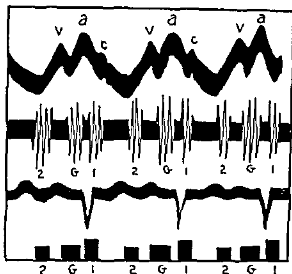


FIG 79 —Illustration of mesodiastolic gallop rhythm in which the gallop sound G is produced by a summation of the third and fourth heart sounds The addition of the two sounds results in a sound sufficiently intense to be definitely audible and still remain properly grouped to produce the rhythm of a galloping horse

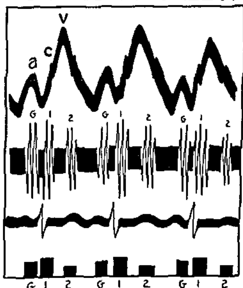


FIG 80 —Illustration of late diastolic or presystolic gallop rhythm in which the gallop sound G, is produced by an accentuated auricular or fourth heart sound

TABLE 5 — BRIEF SUMMARY OF THE CLASSIFICATION AND CHARACTERISTICS OF GALLOP OR "TRIPLE" RHYTHM

Physiologic Nomenclature	*Common Name*	*Heart Sound Responsible*	*Mechanism of Accentuated Sound*	*Time*	*Incidence Per cent*
Presystolic or auricular	Presystolic	4th sound	Auricular systole	Late diastole	27
Rapid ventricular filling	Protodiastolic	3rd sound	Rapid inflow of ventricle	Early diastole	11
Summation (complete or incomplete)	Mesodiastolic	3rd and 4th sounds	Both of above	Middiastole	[illegible]

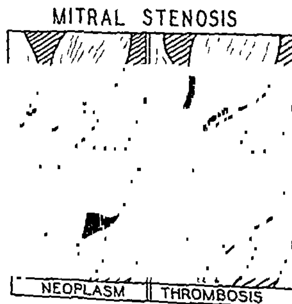


Fig 81 ... may be pro... pitched run...

The Mitral Valve

Mitral Stenosis —The left atrioventricular orifice can be narrowed by inflammation and fibrosis (rheumatic fever arteriosclerosis) of the mitral valve thrombic formation in the left atrium or neoplastic growth in the region of the AV opening (Fig 81) With extreme dilatation of the left

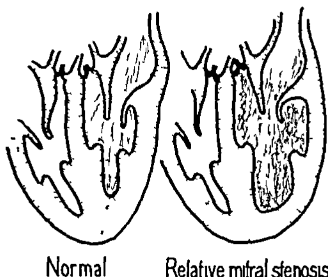


FIG 82 —Illustration showing the production of a relative mitral stenosis with turbulence of flow resulting in a low pitched diastolic rumbling mitral murmur In the dilated heart blood enters through a relatively narrow (actually normal or slightly larger) AV opening into a larger chamber the ventricle This results in turbulence

ventricle and little change in the circumference of the left AV orifice a relative stenosis can develop (Fig 82) Because of the actual or relative narrowing of the left AV orifice blood enters the left ventricle from the left atrium with difficulty and with a consequent turbulence of flow and murmur formation

Turbulence of Flow —The mechanism by which turbulence of flow and sound production occur is shown in figures 83 and 84 The physical nature of these disturbances should be understood by the student before he undertakes the study of murmurs in valvular disease As fluid flows through a tube the friction along the wall of the tube between the flowing fluid and the wall tends to retard the rate of flow of the fluid near the wall The farther the flowing fluid is from the wall of the tube the less friction and the more rapid the rate of flow Therefore fluid tends to flow more rapidly in the center of the tube and less rapidly near the wall being least rapid immediately along the internal surface of the tube The *smoother the surface* of the tube the less the disturbance in flow The more rapid *the rate of flow* the greater the disturbance (Fig 84) It is obvious therefore that any irregularity of the inner surface of the vessel or wide variation in size of the lumen results in turbulence of flow with eddy currents and

(Fig. 84). The more rapid the flow the greater the tur-

the atrium to left ventricle (Fig. 85). The opening is ... the blood flow is not rapid enough to result in turbulence. With stenosis and

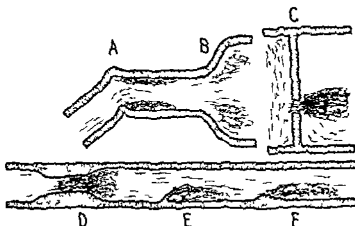
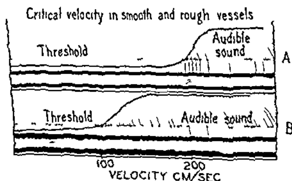


FIG 83 Diagram illustrating the manner in which turbulence results in blood flowing A through sudden angulation of a blood vessel, B into sudden dilatation of a vessel, C through a small defect or opening, D through narrowing of a vessel lumen, E against and around a sclerotic or calcific plaque, and F over a rough or irregular endothelial surface. These factors result in *murmur formation* in the cardiovascular system of man



F[illegible]
for
B
to
ab

narrowing of the mitral valve, turbulence of flow with murmur formation is to be expected (Fig. 85). It is this turbulence of flow which is responsible for the sound vibrations. As mentioned previously, everything else being equal, (1) the narrower the opening and (2) the more rapid the flow of blood the greater is the tendency for turbulence to develop. Therefore it is not surprising that the murmur of mitral stenosis should be late in diastole for it is at this time that the atria contract most vigorously or give their

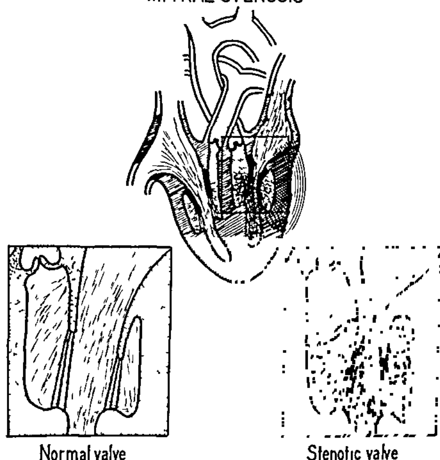
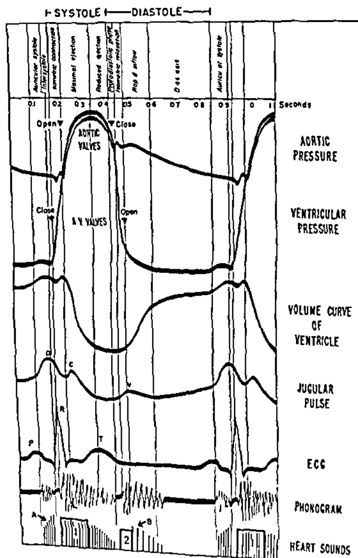


Fig. 85.—Diagram showing the manner in which stenosis of the mitral valve results in turbulence of flow and murmur formation. The normal valve without narrowing and without turbulence is shown for comparison.

last squeeze and eject the last of the blood entering the ventricles. The blood is flowing most rapidly and with greatest force at this moment [illegible]

and thereby fostering still more turbulence of flow. These factors [illegible] lead to a late diastolic or presystolic (not systolic) murmur (Fig. 86).

This murmur is *crescendo* (Fig. 87) in nature because the left atrium contracts progressively and more vigorously, the blood flow accelerates, and the mitral opening progressively narrows. These two factors favor increasing turbulence as the later phase of diastole is reached.



Murmur.—The murmur is *low in pitch, rumbling in quality* and *faint in intensity* because the pressure gradient between the left atrium and left ventricle is relatively small. In other words, the force with which the left atrium ejects blood into the ventricle is relatively small, and turbulence is not apt to be as great as with some of the other types of murmurs (aortic stenosis, mitral insufficiency) in which the forces responsible for the flow of

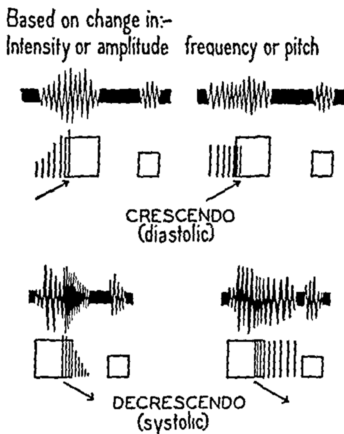


Fig. 87. Illustration of crescendo and decrescendo systolic and diastolic murmurs. Murmurs may be crescendo or decrescendo in pitch or intensity of sound. Various combinations such as decrescendo crescendo, crescendo-decrescendo, etc., are observed.

the blood are relatively great. Furthermore, the opening in the stenotic mitral valve is usually relatively large by comparison with the openings with other valvular lesions.

The relationships of the murmur to the electrocardiogram, jugular pulse record and heart sounds should be thoroughly learned by the student. The principles involved should be obvious and will not be discussed in detail here.

The late diastolic crescendo rumble often builds up rapidly in intensity and ends with a loud "booming" first heart sound (Fig. 88). *The cause of the loud booming first sound is not well understood.*



Fig 88 —Illustration by conventional method of the murmur in which the late crescendo diastolic mitral murmur increases and then terminates suddenly in a loud "booming" first sound

There is often a systolic click (Fig 89) associated with mitral stenosis the mechanism of which is also unknown

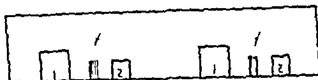


Fig 89 The systolic click encountered in mitral stenosis Its mechanism is unknown It is a high pitched short metallic sound



There may be an *early diastolic decrescendo* murmur in mitral stenosis however (Figs 90 and 91) This again is not surprising when we consider the hemodynamics of the heart for when the left ventricle goes into diastole the intraventricular pressure falls rapidly and the pressure built up in the left atrium from the blood which ... e lungs during ... this difference ... ventricle fills ... this difference in pressure and rate of blood flow decreases the murmur therefore progressively decreases in intensity and is decrescendo in nature (Fig 91)

Murmur.—The murmur is *low in pitch, rumbling in quality* and *faint in intensity* because the pressure gradient between the left atrium and left ventricle is relatively small. In other words the force with which the left atrium ejects blood into the ventricle is relatively small and turbulence is not apt to be as great as with some of the other types of murmurs (*aortic stenosis, mitral insufficiency*) in which the forces responsible for the flow of

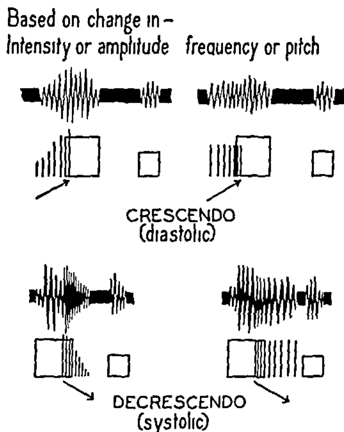


FIG. 87.—Illustration of crescendo and decrescendo systolic and diastolic murmurs. Murmurs may be crescendo or decrescendo in pitch or intensity of sound. Various combinations such as decrescendo-crescendo, crescendo-decrescendo, etc., are observed.

the blood are relatively great. Furthermore the opening in the stenotic mitral valve is usually relatively large by comparison with the openings with other valvular lesions.

The relationships of the murmur to the electrocardiogram, jugular pulse record and heart sounds should be thoroughly learned by the student. The principles involved should be obvious and will not be discussed in detail here.

The late diastolic crescendo rumble often builds up rapidly in intensity and ends with a loud "booming" first heart sound (Fig. 88). The cause of the loud booming first sound is not well understood.

Incidentally the sound tracings of murmurs do not show decrescendo or crescendo characteristics, as is usually diagrammed They merely indicate variations in frequency and amplitude (Figs 86 and 87)

Because these murmurs are relatively low in intensity, they are sharply localized (to an area of 2 or 3 square centimeters) and are heard best near the apex where the left ventricle is nearest the thoracic wall just medial to the apex of the heart *The murmur is often elicited best by bringing the patient's heart near the anterior thoracic wall* This is accomplished by having the patient lie on his left side or lean forward to bring the anterior

a manner similar to that described previously for the murmur



Fig 92 The crescendo-decrescendo mid-diastolic low pitched rumble of mitral stenosis

Etiology —Mitral stenosis is usually the *result of rheumatic fever* It may result from congenital malformations neoplastic growths in the region of the valve intra-atrial thrombotic formation (ball valve thrombosis, which as a museum curiosity may produce complete obstruction to the [illegible] opening) and vegetations of subacute bacterial endocarditis and lupus

insufficiency This Flint murmur (Fig

flowing back into the left ventricle during diastole through the region of the posterior aortic cusp

the leaflet remains partially closed This results in stenosis and interference with blood flow from the left atrium with turbulence of flow and sound or murmur production The murmur usually has the same characteristics as those of relatively mild organic stenosis



Fig 93 The late diastolic low pitched rumbling crescendo Austin Flint murmur Consult the text for details

Quite frequently a patient with mitral stenosis will present both the early decrescendo rumbling murmur and the late crescendo murmur (Fig. 91). Depending upon the cardiac rate, cardiac mechanism and variations in hemodynamics there may be a mid-diastolic, low-pitched, rumbling murmur. This murmur may be crescendo-decrescendo in nature (Fig. 92).

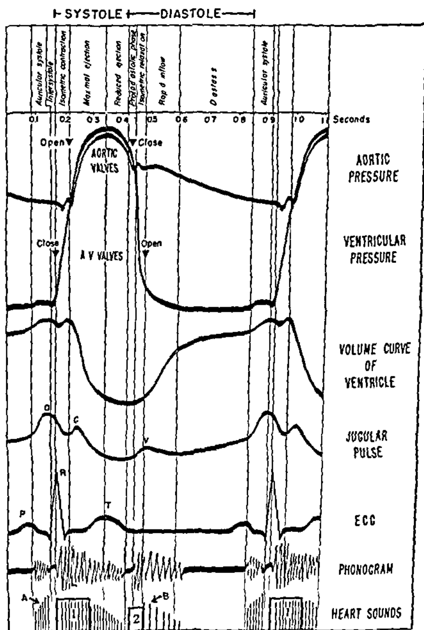


Fig. 91. Illustration of the early decrescendo diastolic murmur (arrow *B*) of mitral stenosis. Consult the text for details.

There often is a sound heard in mitral stenosis called the "opening snap." This is caused by sudden opening of the mitral valves by the relatively high pressure in the left atrium. The sound is not a splitting of the second mitral sound but a third heart sound which precedes the normal third heart sound (Fig. 96).

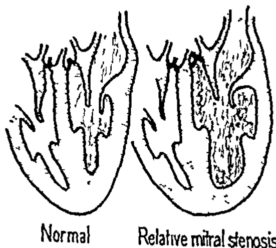


Fig. 95.—Illustration of the mechanism by which dilatation of the left ventricle results in relative mitral stenosis.

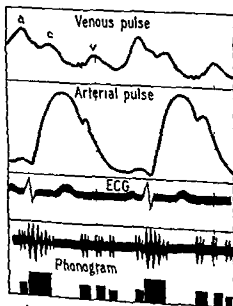


Fig. 96.—Illustration of the opening snap of mitral stenosis. The sounds in order are fourth, first, second, opening snap and third.

Another type of functional mitral stenosis is the so-called *relative stenosis* of left ventricular enlargement (Fig 95). This is not a true stenosis in that there is no interference with or obstruction to the flow of blood from the left atrium into the left ventricle. Because of the dilated and large left ventricle the blood passes through a *relatively* narrow mitral

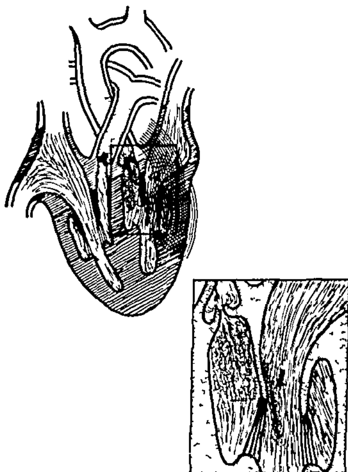
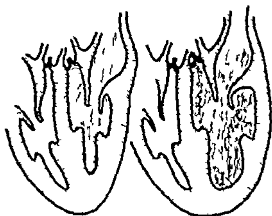


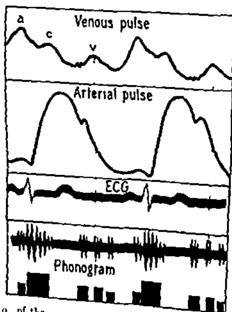
Fig 94.—Diagram showing the mechanism by which aortic insufficiency produces the Austin Flint murmur. The blood regurgitating into the left ventricle from the aorta strikes against the anterior leaflet of the mitral valve. Since the pressure from the aorta is greater than that from the left atrium the mitral valve is held partially closed resulting in a functional type of mitral stenosis.

opening resulting therefore in turbulence of flow and murmur production. Any factor which produces left ventricular dilatation can produce this type of rumbling or low pitched late diastolic murmur. For example any disease that produces strain on the left ventricle, such as diastolic hypertension, coarctation of the aorta, hyperthyroidism, and the like will produce the murmur. The murmur has the same characteristics as those of mild organic mitral stenosis, except that it is less intense and usually occurs only in late diastole.

There often is a sound heard in mitral stenosis called the opening snap. This is caused by sudden opening of the mitral valves by the relatively high pressure in the left atrium. The sound is not a splitting of the second mitral sound but a third heart sound which precedes the normal third heart sound (Fig. 90).



F[illegible] Illustration of [illegible] results in relative [illegible]



F[illegible] Illustration of the opening snap of [illegible] The sounds in order are fourth, first, second, opening snap and third.

There may be other changes in the sounds in mitral stenosis which are usually of two origins (1) The second heart sound is often changed in mitral stenosis Because of the high pulmonary artery pressure, the pulmonary valves close vigorously and the second or pulmonary component of the second heart sound is accentuated This produces a more readily apparent splitting of the second heart sound over the pulmonary valvular area (Fig 97)

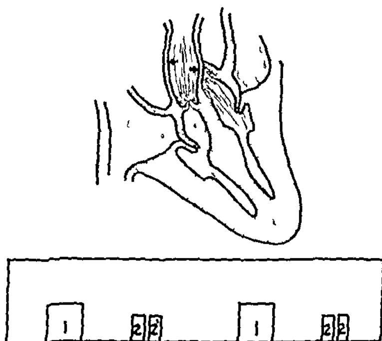


Fig 97 — The increased pressure in the pulmonary circuit (upper diagram of figure) brought about by the mitral stenosis often results in vigorous closure of the pulmonic valve This vigorous closure of the pulmonic and aortic valves produces more readily detectable splitting of the second heart sound (lower part of figure) at the base of the heart

(2) A third or extra sound may be separated from the second mitral sound by more than 0 11 second (mean 0 14 second) in which case it usually represents the beginning of the rumble and is often confused with the physiologic third sound

It is felt that these sounds including the opening snap originate from the mitral valve since they always occur at the summit of the v wave of the jugular pulse tracing and persist during auricular fibrillation The high intra-auricular pressure resulting from the mitral stenosis forces the mitral valve open suddenly when the ventricle goes into diastole

Tricuspid Stenosis — Tricuspid stenosis a rare entity has the same characteristics etiology and mechanisms of production as mitral stenosis It is therefore unnecessary to enter into a detailed discussion of this clinical state and its associated murmurs and thrill The relative positions in the chest for the two murmurs are shown in figure 98

Mitral Insufficiency or Regurgitation—When the mitral valve is insufficient or incompetent it fails to perform its sole function that is to prevent the flow of blood from the left ventricle to the left atrium during ... Normally during systole these leaflets close the ...

left ventricle escapes through this opening ... systole of the ventricle the intraventricular pressure is considerably higher than the intra atrial pressure. These relatively great differences in pressure result in rapid flow of blood usually through a rather small opening between the mitral leaflets. As previously mentioned this results in turbulence of flow with the formation of a high pitched murmur (Fig. 99). Since the murmur is produced during the systolic phase of the ventricle ... heart it is a *systolic murmur*.

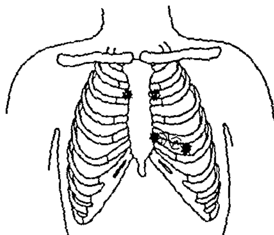


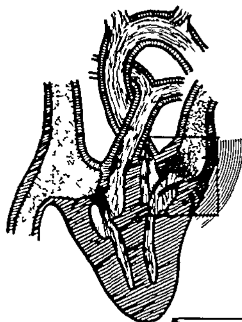
Fig. 98—Topographic diagram of the rel...

The murmur usually occurs early in systole becoming progressively more intense then less intense as systole progresses. The murmur usually starts with the beginning or end of the first heart sound and persists for a variable length of time. It may extend throughout cardiac systole that is to the beginning of the second heart sound.

When the systolic murmur of mitral insufficiency is associated with mitral stenosis there may be a clicking almost metallic sound superimposed on the systolic murmur. This click varies in its position in the systolic phase of the cycle.

The relationship of the murmur to the simultaneous electrocardiogram, venous pulse, and heart sounds should be learned (Fig. 100). Furthermore the reason for these relationships should be mastered to ensure adequate knowledge of the hemodynamic mechanisms responsible for the murmur.

The murmur is heard best near or at the apex of the heart, that is, over the clinical mitral valvular area. The murmur is transmitted, as a rule, laterally to the axillary line. When it is intense, it may be heard over the entire precordium or chest.





[illegible]

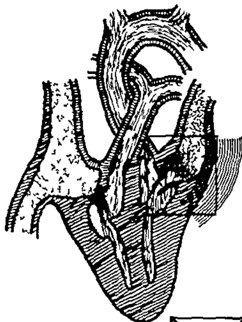
A good rule to remember about the *transmission of murmurs* is that they tend to be transmitted in the direction of flow of the blood which is responsible for the turbulence producing them. Of course, such factors as the nature of intervening tissue and proximity to the thoracic wall also aid in determining the site at which the murmur will be best heard.

The most common systolic murmur at the apical region of the heart is not due to organic heart disease but is a *functional murmur*, that is, one not due to organic valvular disease *itself*. The functional murmurs often have no known cause. Many are associated with systemic or noncardiac infections, many occur in anemic states (*hemic murmur*), and others are associated with cardiac or left ventricular dilatation.

FIG 100 — Illustration of the systolic murmur of mitral valvular insufficiency in relation to other cardiac phenomena

The relationship of the murmur to the simultaneous electrocardiogram, venous pulse and heart sounds should be learned (Fig 100). Furthermore, the reason for these relationships should be mastered to ensure adequate knowledge of the hemodynamic mechanisms responsible for the murmur.

The murmur is heard best near or at the apex of the heart, that is, over the clinical mitral valvular area. The murmur is transmitted, as a rule, laterally to the axillary line. When it is intense, it may be heard over the entire precordium or chest.





which mitral valvular ... The insert shows particularly well mitral valvular leaflets open

A good rule to remember about the *transmission of murmurs* is that they tend to be transmitted in the direction of flow of the blood which is responsible for the turbulence producing them. Of course, such factors as the nature of intervening tissue and proximity to the thoracic wall also aid in determining the site at which the murmur will be best heard.

muscle function and laxness of the musculature resulting in increased muscle sound production.

Etiology.—The principal causes of organic mitral valvular dysfunction with incompetence or insufficiency are rheumatic fever, arteriosclerosis, congenital defects, and vegetative growths on the leaflets (subacute bacterial endocarditis, lupus erythematosus disseminatus). The functional

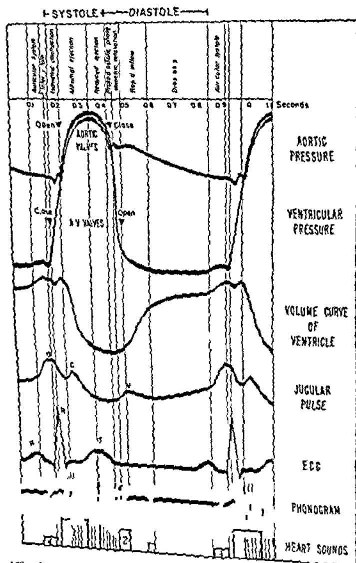


Fig. 102. Illustration of the systolic aortic murmur of aortic stenosis in relation other cardiac phenomena.

The *hemic murmur* is said to be produced by the reduced viscosity of the blood associated with the low hemoglobin erythropenia and associated low concentration of plasma proteins. Fluids low in viscosity are more susceptible to turbulence of flow. In anemia the blood of low viscosity becomes turbulent as it passes around the papillary muscles and chordæ tendineæ. Such a concept is highly theoretic however, and awaits proof. The most likely cause of the hemic murmur is dilatation of the left ventricle produced by insufficient oxygen supply.

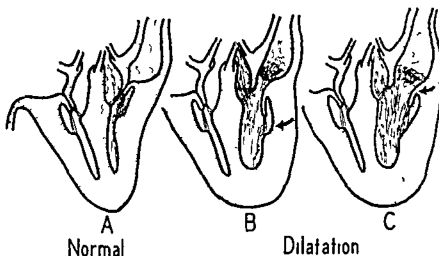


FIG. 101. Diagrammatic representation of the mechanism by which dilatation of the left ventricle results in mitral insufficiency. Part A shows the mitral valve of the normal heart properly closed and retained in the proper position by the chordæ tendineæ. When the ventricle dilates the walls expand centrifugally displacing the papillary muscles and attachments of the chordæ tendineæ. The fibrous chordæ tendineæ do not stretch in this process, therefore during the early period of systole they hold the mitral valves partly open (Part *B*) since they are not long enough to allow the mitral leaflets to approximate properly. This is the most important factor responsible for functional mitral insufficiency. The valve remains insufficient for a long period of the systolic phase as shown in the almost completely evacuated ventricle of Part *B*. There is an additional slight dilatation of the mitral ring (Part *C*) whenever the ventricles dilate.

The functional systolic mitral murmur of mitral insufficiency *produced by dilatation of the left ventricle* is probably attributable to the following factors:

1. When the left ventricle is dilated the papillary muscles tend to move away from the valvular leaflets. With the chordæ tendineæ unable to ... iplete approxima- ... factor ... ing dilates thus enlarging the cross sectional area of the orifices. The leaflets remain the same size and therefore are unable to close the mitral orifice completely.

3. A third factor contributing to the murmur of left ventricular dilatation is its "flabby" nature. There is the associated impairment of papillary

The murmur of aortic stenosis is transmitted into the arteries of the neck since the blood is flowing in that direction when it is produced. The murmur is of the decrescendo type, for the pressure gradient (difference between intraventricular and intra-aortic pressures) is greatest early in systole. As the aortic pressure increases and this gradient becomes less the rate of blood flow through the aortic opening progressively decreases and the murmur accordingly decreases in intensity (Fig. 102).

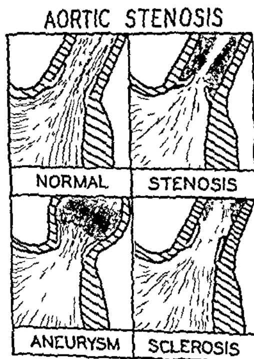


Fig. 104. The common factors responsible for an aortic systolic murmur. Because …

Aortic stenosis is usually produced by the … fever … [illegible]

… openings if they are to function mechanically as efficiently as possible … at the aortic and pul… (Fig. 105)

insufficiency associated with left ventricular dilatation results from clinical states which produce strain on the left ventricular musculature (diastolic hypertension aortic stenosis aortic insufficiency) and acute myocarditis (rheumatic Fiedler's diphtheritic acute nephritic etc)

It is well to remember that the lesion of organic stenosis not only interferes with blood flow through the mitral opening into the ventricle but also interferes with closure of the valve during systole *Therefore* mitral stenosis is almost invariably accompanied by a systolic murmur of mitral insufficiency

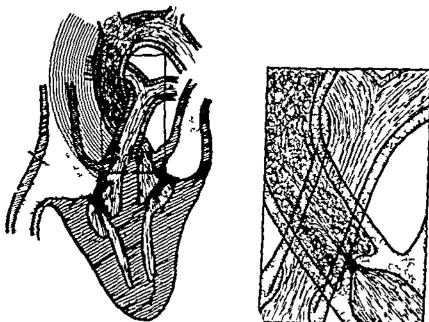


Fig 103 —Diagram of the mechanism of the turbulence of flow produced by aortic stenosis This is responsible for the systolic murmur It is during systole that blood flows through the aortic valve into the aorta

The Aortic Valve

Aortic Stenosis As a result of narrowing (stenosis) of the aortic orifice a systolic high pitched murmur is found over the aortic valvular area (Fig 102)

When the left ventricle goes into systole and the pressure within the ventricle exceeds that in the aorta blood flows out of the ventricle into the aorta If there is thickening of the aortic cusps or narrowing of the opening the rapidly flowing blood passing the constricted region becomes turbulent (Fig 103) with the resultant formation of murmurs Since blood flows out through the aortic opening at a great speed and under high pressure the murmur is high pitched When the stenosis is great or the opening small the turbulence is more vigorous and the murmur is harsh and loud Figure 104 shows the turbulence of flow in (1) *aortic stenosis* (2) *aortic plaques* and (3) *aortic aneurysm*

aortic cusps and ring such as syphilitic valvulitis result in incompetence of the aortic valve so that when the ventricle is in diastole and the intra ventricular pressure level is lower than that in the aorta the aortic cusps fail to support the blood and it therefore regurgitates rapidly into the left ventricle past the partially closed aortic cusps Turbulence of flow therefore results and a murmur is produced (Fig 107) The murmur

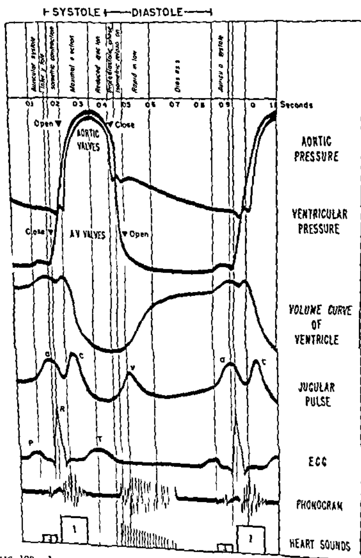
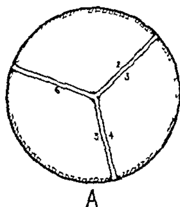
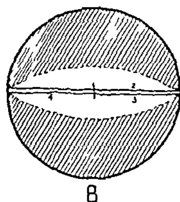


Fig 106 relation to the implications

The aortic second sound is often less intense because of the lower pressure and reduced *volume of blood pumped into the aorta.* This occurs because of the obstruction to blood flow offered by the stenosis.

A systolic murmur of a relative or functional aortic stenosis is produced by dilatation of the *root of the aorta or projection of an arteriosclerotic* calcified plaque into the aortic lumen (Fig. 104). Congenital atresia of the *aorta (coarctation of the aorta)* will also produce the same findings





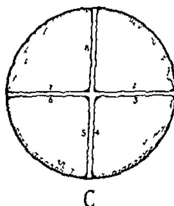


Fig. 105. Diagram to show why three cusps [illegible]
[illegible]

described previously for aortic stenosis except that the murmur is most intense at a different point on the chest. True aortic stenosis is more likely to have an associated thrill than are the other states which produce a systolic aortic murmur.

Aortic Insufficiency Regurgitation or Incompetence. The murmur of aortic regurgitation is diastolic in time (Fig. 106). This is logical when considered from the standpoint of cardiac hemodynamics and the anatomic defect. When the left ventricle is in diastole the aortic valve supports the blood in the aorta *(under high pressure) and prevents it from flowing* back into the ventricle which is under low pressure. Diseases of the

The duration of the murmur varies usually extending throughout the entire diastolic period . . . . of flow of the blood which
The murmur . . . is
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heart (Fig 108)
or the apical region of the heart and is often readily heard in all three areas The murmur may be pronounced at any one of these areas At

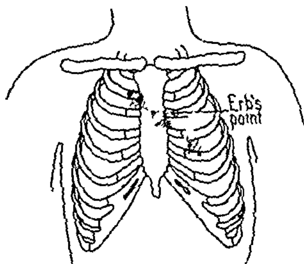


Fig 108 Topographic distribution of the diastolic murmur of aortic regurgitation It is most intense over the aortic area then Erb's point and then at the apical region as it is transm . . .
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at th sound produ ng vibration

Erb s point (third and fourth left intercostal parasternal area) the heart is near the thoracic wall and the turbulent blood is relatively near the chest piece of the stethoscope

The rumbling *Austin Flint* murmur (a functional diastolic murmur of

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is high-pitched and blowing in character because the opening in the aortic valve *is usually relatively small and the flow back into the left ventricle* is rapid. The murmur is *decrescendo* in character because the difference in pressure (or pressure gradient) is greatest when the ventricle first goes into diastole. At that time the intraventricular pressure is near zero and the pressure in the aorta is relatively high. As the ventricle fills and

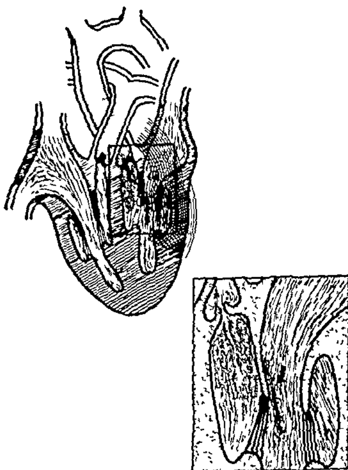


FIG. 107.—Diagram of the mechanism of the Austin Flint murmur. The pressure of the blood leaking back from the aorta into the left ventricle strikes against the anterior leaflet of the mitral valve held partially closed, thereby resulting in functional mitral stenosis.

its pressure rises, the pressure in the aorta progressively falls, thus there is a progressive decrease in the rate of flow of blood back into the ventricle and consequently a progressive diminution in the intensity of the murmur through diastole.

The duration of the murmur varies usually extending throughout the entire diastolic period

The murmur is transmitted in the direction of flow of the blood which produces the murmur Therefore it is transmitted toward the apex of the heart (Fig 108) The murmur is heard best at the aortic area *Erb's point* or the apical region of the heart and is often readily heard in all three areas The murmur may be pronounced at any one of these areas At

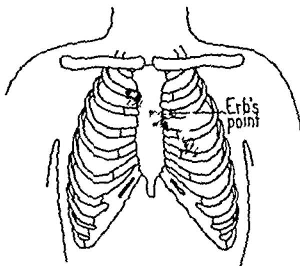


Fig 108 —Topographic distribution of the diast...

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result systolic murmur and a diast lic murmur of a blowing or high pitched ... to the insufficiency there

type, a so-called "to-and-fro" murmur. This is more likely to occur in syphilitic aortic regurgitation. Rheumatic fever and arteriosclerosis result in predominance of stenosis (systolic murmur).

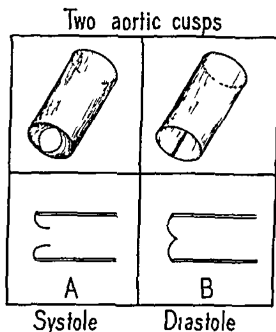


FIG 109.—When there are two aortic cusps rather than three (a congenital anomaly) stenosis results but insufficiency is not likely to develop. The valves cannot open completely during systole (Part A) producing stenosis and a systolic murmur but they close properly (Part *B*). In organic disease of the cusps both stenosis and insufficiency are usually associated for the cusps do not function properly during systole and diastole. This is an important point of differentiation between acquired and congenital aortic stenosis. The same congenital defect may occur in the pulmonary valve.

A congenital aortic defect (two aortic cusps) usually results in only a stenotic murmur (systolic) and no diastolic murmur: these two cusps fail to open completely but do close completely (Fig 109). This is usually of diagnostic importance.

Functional aortic insufficiency with the typical high-pitched blowing murmur may occur in association with *acute myocarditis* or with acute *aortitis*, in which the aortic ring or region of attachment of the cusps of the aortic orifice is dilated. This results in an increase in the cross-sectional area of the aortic valvular orifice. The cusps are therefore unable to close the opening completely during diastole and blood regurgitates into the left ventricle, resulting in the production of a murmur. *Severe diastolic hypertension* may also produce this murmur. The high diastolic pressure distends the root of the aorta, increasing the cross sectional area of the aortic orifice and producing aortic valvular incompetence.

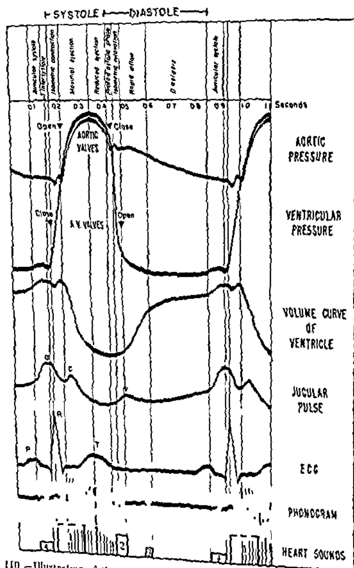


FIG. 110.—Illustration of the murmur of pulmonary stenosis with its relation to other cardiac phenomena

The Pulmonary Valve

Pulmonary S...

usually due to congenital atresia scarring from rheumatic fever, or vegetative growths (bacterial endocarditis or lupus erythematosus disseminatus) on the cusps

In pulmonary stenosis the pulmonary second sound is often greatly reduced in intensity This is due to the reduced pressure in the pulmonary artery resulting from the decreased volume of blood emptied into the pulmonary artery The volume output and pressure are reduced by the obstructing stenosis

Fig 111 —Illustration of the murmur of pulmonary valvular insufficiency

pulmon ... sible for the murmur may reside in the ... forward until the anterior surface

The murmur is a soft ... ... extends *throughout systole* intensity which occurs early in systole ... and tends to disappear on deep inspiration. It has been found to occur in about 85 per cent of normal medical students





Pulmonary Insufficiency Regurgitation or Incompetence—This is a rare cause of murmurs. The murmur is diastolic in time (Fig 111) and it may occur in congenital defects of the pulmonary valve or in organic rheumatic disease. It has essentially the same characteristics as the murmur of aortic insufficiency except that it is heard over the pulmonary valvular area and is transmitted downward along the left margin of the sternum

usually due to congenital atresia, scarring from rheumatic fever, or vegetative growths (bacterial endocarditis or lupus erythematosus disseminatus) on the cusps

In pulmonary stenosis the pulmonary second sound is often greatly reduced in intensity. This is due to the reduced pressure in the pulmonary artery resulting from the decreased volume of blood emptied into the pulmonary artery The volume output and pressure are reduced by the obstructing stenosis.

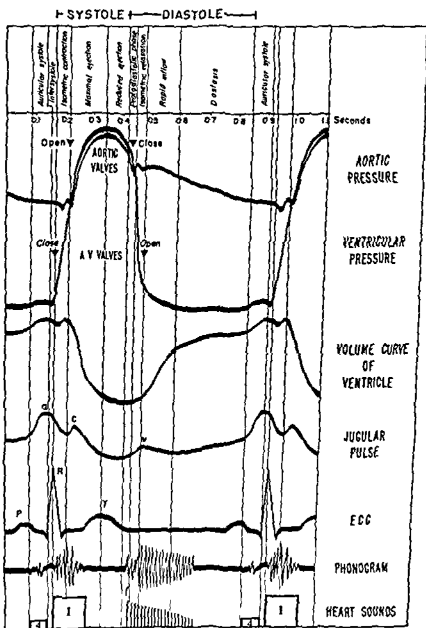


FIG 111 —Illustration of the murmur of pulmonary valvular insufficiency

artery into the right ventricle with turbulence of flow and murmur production (Fig 112) A good example of this type of functional diastolic pulmonary valvular murmur is seen in *mitral stenosis* Because of the narrowing of the mitral orifice blood passes into the left ventricle with difficulty and therefore accumulates under high pressure in the left atrium pulmonary veins and capillaries and arterial system This results in dilatation of the pulmonary ring and functional regurgitation This particular murmur is known as the *Graham Steell murmur*

The Tricuspid Valve

Stenosis of the tricuspid valve is rare The etiology, hemodynamics mechanisms and characteristics of the murmur are essentially the same as for mitral stenosis (Fig 113) The main difference is the location of the murmur which is just to the left of the sternum at the level of the fifth rib or intercostal space The jugular pulse record aids in the diagnosis of this condition

Tricuspid Insufficiency Regurgitation or Incompetence —This is a common syndrome when the functional type of tricuspid insufficiency is included in the consideration The entity on an *organic basis* is not uncommon however The murmur is systolic in time (Fig 114) and has the same characteristics and mechanisms described for mitral insufficiency except that it is located over the *tricuspid area* and is *transmitted* to the right in many instances

The configuration of the jugular venous pulse tracing is rather characteristic (Fig 114) The c wave is greatly exaggerated for when the right ventricle goes into systole the incompetent tricuspid valve permits blood to regurgitate into the right atrium veins of the neck inferior vena cava and hepatic veins (with resultant *pulsating liver*) (Fig 115) This regurgitated blood produces an increase in the v wave to an extent pro-

any other murmur de-
... may be great enough to produce a palpable vibration (thrill) of the overlying tissues The thrill is usually most intense over the area at which the murmur is most intense

Congenital Cardiac Anomalies

The number and types of cardiac anomalies are far too great to present completely in this type of monograph Once the student has learned the fundamental principles of m ... modynamics he ... few of the more

... frequently the interventricular ... is to close completely, resulting in a *Roger s murmur* It is usually

Functional pulmonary insufficiency with the soft blowing, high-pitched diastolic murmur is encountered rather frequently. It is seen in diseases associated with *pulmonary arterial hypertension*. This results in distention of the pulmonary artery and pulmonary ring with an increase in the cross sectional area of the orifice. The cusps are therefore unable to close the opening during diastole of the right ventricle, since they do not enlarge correspondingly. This results *in regurgitation of blood from the pulmonary*

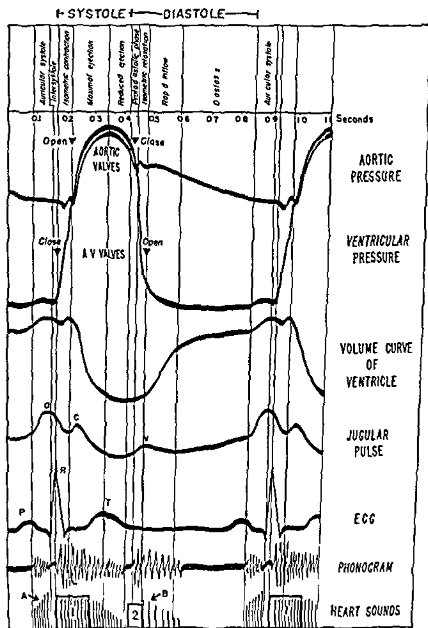


FIG. 113.—Illustration of the murmur of tricuspid stenosis

arteries into the right ventricle with turbulence of flow and murmur production (Fig 112) *A good example of this type of functional diastolic pulmonary valvular murmur is seen in mitral stenosis* Because of the narrowing of the mitral orifice, blood passes into the left ventricle with difficulty and therefore accumulates under high pressure in the left atrium pulmonary veins and capillaries and arterial system This results in dilatation of the pulmonary ring and functional regurgitation This particular murmur is known as the *Graham Steell murmur*

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any other murmur de-
... may be great enough to produce a palpable vibration (thrill) of the overlying tissues The thrill is usually most intense over the area at which the murmur is most intense

Congenital Cardiac Anomalies

The
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Not infrequently the interventricular ... fails to close completely resulting in a *Roger's murmur* It is usually

due to failure of closure of the *membranous portion* of the interventricular septum (Fig 116). The murmur is systolic in time because the pressure within the left ventricle exceeds that within the right, and therefore blood *flows from the left ventricle to the right ventricle* when the two enter systole simultaneously. Since aerated blood is mixing with reduced hemoglobin, *no cyanosis* results Only when the septal defect is large (cor triloculare biauriculare) and there is churning with more thorough mixing

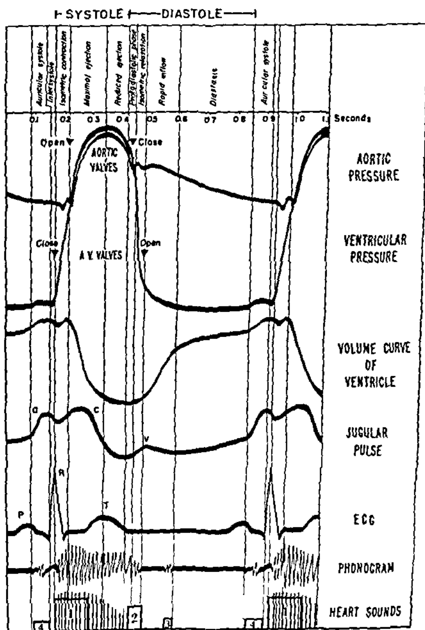


Fig 114 —Illustration of the murmur of tricuspid insufficiency

This is relatively rare ... of blood from the two ventricles is then ... In general the ... compatible with only a short life (hours to weeks) ... (Fig 117) This murmur ... murmur described previously

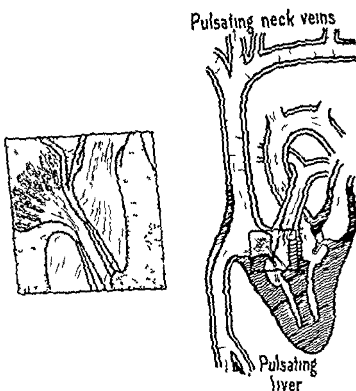


Fig 118 —Diagram ... atrium superior ...

The murmur is *most intense* near the left margin of the sternum at the level of the fourth chondrosternal articulation and intercostal space It is best heard above the *tricuspid valvular area* This area is often referred to as Roger's area (Fig 118) The transmission of the murmur varies with its intensity if intense it may be transmitted over the entire precordium and often to the posterior thorax A *thrill* is often felt with the murmur being most intense over the region where the murmur is loudest

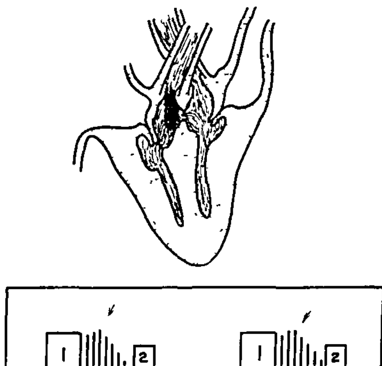
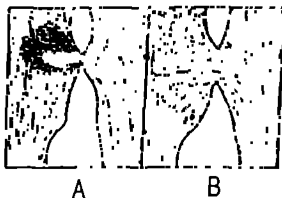


FIG 116 —Illustration of the manner in which a defect in the interventricular septum results in turbulence of flow and a systolic murmur Since the pressure is greater in the left ventricle than that in the right, aerated blood mixes on the right side of the heart and no cyanosis occurs The murmur, *Roger's murmur*, is shown at the lower part of the figure The defect is most frequent at the superior or membranous portion of the interventricular septum



Patent Interauricular Septum.—This is a common entity when patency of the *foramen ovale* is included in the group. As a rule the opening is so small that no murmur can be detected. This is also explained by the pressure differences between the two atria being too small to result in sufficiently rapid flow of blood through the opening to produce turbulence.

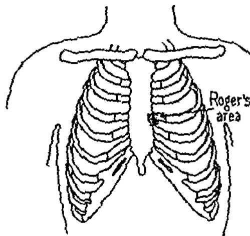


Fig 118 The point (shaded area) on the chest where the Roger's murmur of a patent interventricular septum is most intense is known as Roger's area

When the opening is large enough, a presystolic-systolic murmur results (Fig 119). The flow is from the left to the right atrium. The murmur has essentially the same characteristics as the other systolic murmurs previously described. Many clinicians are of the opinion that the systolic murmur of atrial septal defect results from the pulmonary vascular plethora and therefore is produced in the engorged pulmonary arteries and not at the septal defect.

It is loudest over the midsternal region at the level of the second intercostal space or in some cases just to the left of the midsternal line (Fig 120). It is not *transmitted* widely unless it is intense and it tends to be transmitted into the left infraclavicular region, *i.e.*, in the direction of blood flow in the pulmonary artery. A *thrill* is often associated with the murmur, being most intense in the area where the murmur is loudest.



Fig 119.—The presystolic-systolic murmur of patent interatrial septum

This defect is not associated with cyanosis as aerated blood flows into the right atrium. There is cyanosis when the septal defect is extremely large and much mixing and churning of blood take place (cor triloculare biventriculare).

Pulmonary Stenosis.—This has been discussed under the general presentation of pulmonary valvular disease.

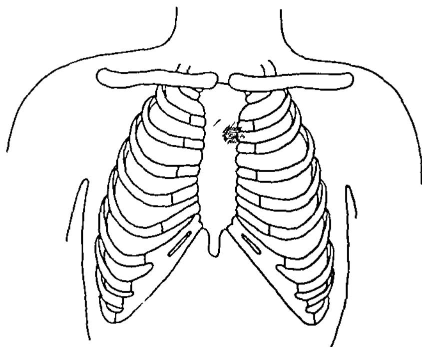


Fig. 120. Topographic illustration of the point (shaded area) at which the murmur of interatrial septal defect is most intense.

Aortic Stenosis and Coarctation of the Aorta. These defects have been discussed under aortic valvular disease in general.

Combination Anomalies.—There are frequently combinations of anomalous defects of the heart as for any organ system. When this is true various defects will present their respective murmurs. A good example of this is *tetralogy of Fallot* in which the murmurs of pulmonary stenosis and patent interventricular septum occur together. The student should be alert to such combined anomalies when listening to the heart. These will be discussed further with the clinical presentation of congenital cardiac disease.

Patent Ductus Arteriosus.—This is a fairly common congenital defect which has interesting associated murmurs. There is a characteristic *machine murmur* (Fig. 121 *B*) which is simple to understand if one is aware of the anatomic defect present and the respective pressure levels. In the typical instance there is a continuous murmur because the pressure

in the aorta is at all times higher than that in the pulmonary artery (Fig
... During ventricular systole the pressure in the aorta is much greater

louder than the diastolic portion ...
rapidly from aorta to pulmonary artery because the pressure difference is

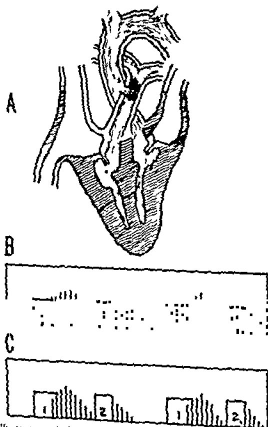


Fig 121 Illustration of the mechanism (Part 1) of the turbulence of flow in congenital patency of the ductus arteriosus ...

smaller ...
the pressu...
ary valve ...

The murmurs are most intense in the second intercostal space slightly to the left of the sternum They may be transmitted widely over the upper portion of the left hemithorax and precordium

A continuous *thrill* with systolic exacerbations may be associated with the murmur. There is no cyanosis since oxygenated hemoglobin is being added to reduced hemoglobin in the pulmonary circulation and no reduced hemoglobin enters the aorta and general circulation.

Not infrequently only a *systolic murmur* is present or two *separate and distinct* systolic and diastolic *murmurs* are heard (Fig. 121 C). The diagnosis is difficult when this is true. It is particularly apt to occur early in life and usually occurs when the opening is small or the duct enters the aorta or pulmonary artery at an extremely acute angle. At times there may be only a systolic murmur.

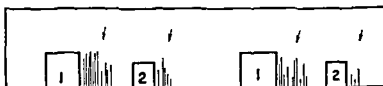


Fig. 122.—Illustration of the systolic and diastolic sounds of a pericardial friction rub.

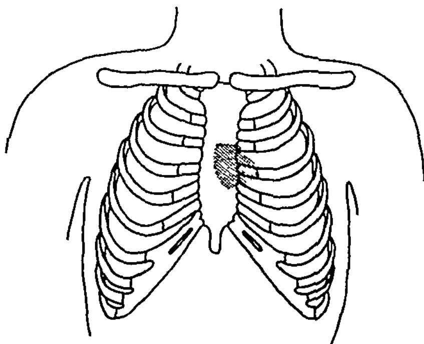


Fig. 123.—Topographic illustration of the most likely location of a pericardial friction rub.

Pericardial Friction Rub

As a result of inflammation of the pericardium its visceral and parietal surfaces are roughened. This roughening results in a considerable amount of friction between the epicardium and pericardium during systole and

diastolic movements of the heart This in turn results in sound vibrations or a *friction rub* (Fig 122) The sound is intensified by pressure over the precordium It is usually most intense near the basal region of the heart at the level of the third or fourth intercostal spaces near the left margin of the sternum (Fig 123) The intensity of the friction rub varies with respiration but is not dependent upon it as is a pleural friction rub Obviously, the drier the pericardial sac the louder is the friction rub When the epicardial and pericardial surfaces are *separated* by pericardial fluid the rub disappears

Miscellaneous Extracardiac Sounds Heard Over the Heart

There are many sounds heard over the precordial region which are *extracardiac* in origin One should become thoroughly acquainted with them in order to avoid the error of considering them significant Many normal individuals have been made to pay extra premiums or actually

[illegible]

[illegible] consider all of the variations of these extracardiac sounds and the many diversified explanations concerning their origin only a few will be considered

Xiphosternal Crunch.—The term xiphosternal crunch has been employed as a general term to include most if not all of the extracardiac sounds This term is not satisfactory since it implies a mechanism for the sounds without such a mechanism ever [illegible]

[illegible]

such as vibrations of the chorda [illegible] the thoracic wall and cardiac [illegible] of them seem to be produced [illegible] the xiphosternal articulation [illegible] caused by movement of the articulation on respiration by the heart pressure over the sternum etc It has been estimated that these extracardiac sounds may be heard in a little over 20 per cent of normal young persons

Cardiorespiratory Sounds Cardiorespiratory sounds are frequently heard These sounds are produced by the beating heart [illegible] the [illegible]

[illegible]

[illegible] inspiration or complete expiration the sounds are altered greatly or may even be eliminated This is an important diagnostic test for identifying them These sounds may be considerably different in character varying from a *blowing murmur* to high pitched *whistles* They usually occur during cardiac systole but may be heard during any phase of the cardiac cycle varying a great deal with the cycle

Pneumothorax is often associated with a peculiar snapping or cracking sound referred to as a pleuropericardial knock (Fig 124)

A continuous *thrill* with systolic exacerbations may be associated with the murmur. There is no cyanosis, since oxygenated hemoglobin is being added to reduced hemoglobin in the pulmonary circulation and no reduced hemoglobin enters the aorta and general circulation.

Not infrequently, only a *systolic murmur* is present or two *separate and distinct*, systolic and diastolic, *murmurs* are heard (Fig. 121, *C*). The diagnosis is difficult when this is true. It is particularly apt to occur early in life and usually occurs when the opening is small or the duct enters the aorta or pulmonary artery at an extremely acute angle. At times there may be only a systolic murmur.

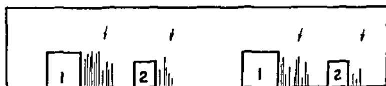


Fig. 122.—Illustration of the systolic and diastolic sounds of a pericardial friction rub.

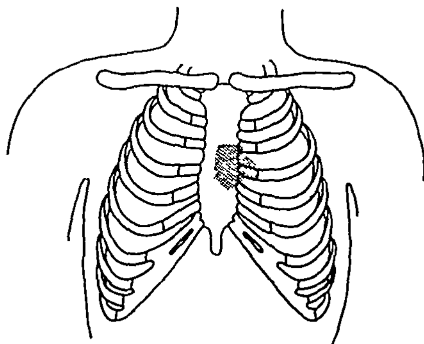


Fig. 123.—Topographic illustration of the most likely location of a pericardial friction rub.

Pericardial Friction Rub

As a result of *inflammation* of the pericardium its visceral and parietal surfaces are roughened. This roughening results in a considerable amount of friction between the epicardium and pericardium during systolic and

1 **Systole en echo** is a faint sound heard during diastole of the ventricles due to contraction of the atria (Fig 126) The faint or low intensity auricular sound suggests an echo



Fig 126 —In complete A-V nodal block the frequently occurring *faint atrial sounds* are [illegible] but definitely audible during the long periods between the ventricular contractions The sound resembles an echo hence the term "systole en écho" The small black blocks at the top of the figure indicate auricular activity

2 Occasionally by chance the mitral first sound is reinforced by an auricular contraction producing a loud sound referred to as **"bruit de canon"** (Fig 127) This may also be due to the fact that the valves were held wide open



Fig 127 —Diagram showing the manner in which simultaneous contraction of the atria and ventricles results in a loud sound "bruit de canon" because of the summation of the fourth and first heart sounds *The loud sounds are indicated by the first sounds* with a broad black line increasing *their height* The small black blocks at the top of the figure indicate auricular activity

3 Irregularly *occurring third and fourth* heart sounds *resemble* a splitting of M_2 and M_1

Auricular Fibrillation —Because of the *irregularity* the first and second sounds occur at irregular intervals and because of the variations in the *force* of ventricular contractions they vary in intensity There may be groupings in threes or fours (Fig 128) Since the atria do not contract there is no fourth heart sound



Fig 128 Illustration of the manner in which the weak ventricular cont[illegible]

Mediastinal Emphysema.—Mediastinal emphysema, regardless of the origin of the air (usually pulmonary, however), is associated with many bizarre crackling sounds. These sounds are produced by the beating heart which displaces the air into the mediastinal tissues, producing the crackling noises (Fig. 125).

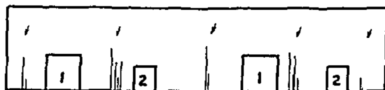


FIG. 124.—Systolic and diastolic high pitched loud "knock" encountered occasionally in pneumothorax. These sounds are *extracardiac* in origin.



FIG. 125. Illustration of the short high pitched crackling sounds of mediastinal emphysema. These sounds are extracardiac in origin and are produced by the rotating and twisting heart as it presses against the bubbles of air in the mediastinum.

Hydropneumopericardium.—Hydropneumopericardium may be associated with a splashing or gurgling sound as the beating heart stirs the fluid and air.

There are many other types of extracardiac sounds which may confuse the listener if he is not alert and observant. These should be kept constantly in mind.

Variations in Heart Sounds Due to Variations in Activation of the Ventricular Musculature

From the foregoing it is obvious that the heart sounds will vary in their timing if there are variations in the timing of the contractions of the various chambers of the heart. A few examples are indicated in the following paragraphs. There are many more, all of which should be predictable by the student.

Bundle Branch Block.—*This has been discussed in detail previously.* Since the ventricles contract asynchronously, the sound produced by the mitral and tricuspid valves must occur at different times.

Complete Atrioventricular Block.—With complete blocking of the AV node, the heart *sounds vary a great deal depending upon the relative* sequence of contraction of the atria and ventricles. Some of the many possibilities are:

Chapter 4

THE COMMON TYPES OF HEART DISEASE

It is not intended in this compendium to present fully, or to discuss partially *all* of the types of cardiac disease. It is planned to introduce the student to the most common forms of heart disease and to present briefly their essential features. There are several good monographs and other publications from which more details can be obtained. The discussion to follow, therefore, cannot be considered encyclopedic.

Disease of the heart is common, even more than is generally realized by the average physician. Furthermore, no one need accept such a diagnosis with a *fatalistic* point of view, since most heart disease is reversible. Unfortunately, however, many types still remain irreversible, and it is these which impress the physician and layman most. A patient with an irreversible heart disease can receive much benefit from proper management. For a physician to administer proper therapy he must have a thorough knowledge of cardiac anatomy, physiology, and pharmacology, as well as the clinical manifestations of the *normal* and *abnormal* heart. Many unfortunate experiences in heart disease are due to poor management. Every student should make a concerted effort to learn cardiology well, for heart disease is an everyday problem in all fields of medicine, regardless of the specialty.

Prior to a discussion of the most frequently encountered types of heart disease, the term Heart Disease should be defined in light of the usage employed throughout this compendium.

Heart disease is defined as any cardiac dysfunction which disturbs the patient in any way. The word *disease* literally means lack of ease or uneasiness.

Obviously, such a definition includes not only *organic* cardiac disease but *functional* cardiac states as well. It includes not only a disturbance in cardiac reserve or inability to supply the tissues of the body with a sufficient amount of blood but

turbances of even a mild sort

bidity or mortality. The pati

doubt about the degree to wh

who may even refrain from

... important because today people ... younger and more of them therefore die of cardiovascular disease. The influence of present-day methods of rapid communication, announcing

Premature Contractions and Pulsus Alternans.—Variations in the heart sounds in these conditions are discussed in Chapter 5 Illustrations (Figs 129 and 130) are repeated here for convenience As an exercise, the characteristics of the sounds should be studied and drawn by the student for many types of cardiac irregularities

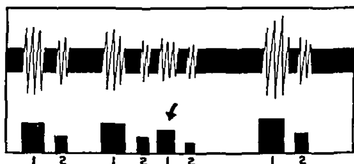


FIG 129 —Variations in number intensity and temporal relations of the heart sounds in this illustration are easily understood if the hemodynamic principles are employed The premature contraction occurs early or prematurely, it is therefore associated with sounds which appear early Since the contraction is weak, the sounds are less intense (noted by arrow) than the regularly appearing ones Because of the compensatory pause, the ventricles then have an opportunity to fill a great deal, therefore the next regularly appearing sounds are loud or intense With greater filling and stretching of the ventricular muscle, the contraction is forceful (Starling's law of the heart) and the associated sounds are intense Consult Chapter 5 for details

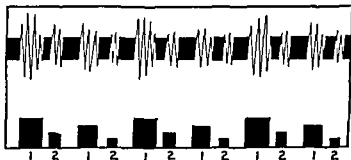


FIG 130 —The alternate variations in intensity of the heart sounds in pulsus alternans The loud sounds occur with the stronger contractions of the ventricles and the less intense sounds with the weaker contractions *The sounds occur on time Consult* Chapter 5 for details

common in extracardiac infections is usually reversible and therefore is of little concern as a residual entity to the patient or his physician. Nevertheless, injury to the heart, usually the myocardium, is common, being almost invariably present in severe infections with intoxication, and should not be neglected.

Types of Infections

Infections which are most prone to produce cardiac damage are the severe pyogenic infections (streptococcal, staphylococcal, meningococcal, pneumococcal) with severe toxemia. Any infection, acute or chronic, may injure the heart. The damage may be so subtle as to escape all but the most careful and meticulous clinical study. Because the injury is so likely to occur in infections, the heart should be studied in all such patients, especially if facilities permit. Injury may be associated with such extracardiac infections as streptococcal follicular tonsillitis, pyogenic paranasal sinusitis, coryza, furunculosis, pneumonia, meningococcic meningitis, childhood infections such as scarlet fever, diphtheria, local infections, and so on. The viral and rickettsial diseases may also damage the heart.

The previously mentioned infections injure the myocardium through the medium of circulating toxins liberated by the infecting agent or microorganism. In many instances microorganisms may infect the heart directly, lodging in the endocardium, pericardium or myocardium. Many instances of cardiac injury occur as a result of direct involvement by diseases which may be due to infections, for example, rheumatic fever, lupus erythematosus disseminatus, etc. Rheumatic fever will be discussed separately.

ACUTE MYOCARDITIS

Since myocardial inflammation or injury is the most common type of cardiac damage associated with infections, it is discussed first. The damage may follow any type of infection, protozoal, bacterial, fungal or viral. The agent usually injures the heart by toxins which reach the myocardium by w[illegible] In some instances the organism ma[illegible]

The [illegible] mild forms the mu cle [illegible] damage being manifested physiologic [illegible] y reveal only early degenerative changes, such as hyaline and hydropic degeneration. When the damage is more extensive there may be fragmentation of the muscle fibers with hypertrophy and at times focal areas of necrosis such as are seen in diphtheria. There may be a slight accumulation of inflammatory cells with relatively mild injury, or there [illegible]

[illegible]

[illegible] variable extent [illegible] ged only slightly or to an im

the sudden cardiac deaths of relatives friends and associates of about the patient s age who were in apparent good health is not to be underemphasized Such announcements result in mental anguish and fear It is necessary therefore in the management of patients in the clinic not to restrict the use of the term heart disease to irreversible condition It will also prevent the average physician from creating a cardiac neurosis as a result of careless speech and *inadequate* evaluation of the cardiac and mental status of a patient

INCIDENCE OF HEART DISEASE

It is impossible to obtain adequate data about the incidence of heart disease especially in its broad sense throughout the entire world For that reason the list of causes for the most frequent types of heart disease presented below is essentially an impression Many observers may want to change the order but this is of no real consequence for the moment especially *in view of the great variations in incidence of heart disease from one section* of a nation to another or from one continent to the next For convenience of discussion it is considered that the most common causes of heart disease for the *entire world* in descending order of incidence are as follows

1 Infection
2 Anemia
3 Malnutrition
4 Psychoneuroses
5 The aging process (arteriosclerosis)
6 Hypertension
7 Syphilis
8 Rheumatic fever
9 Thyroid diseases
10 Congenital anomalies
11 Miscellaneous group of diseases

The incidence of heart disease in the *United States* in descending order would be essentially as follows

1 Infection
2 Anemia
3 Psychoneuroses
4 The aging process (arteriosclerosis)
5 Hypertension
6 Rheumatic fever
7 Malnutrition
8 Syphilis
9 Thyroid diseases
10 Congenital anomalies

It is well to consider that in many instances several of the causes of heart disease are acting at one time For example infection anemia and malnutrition are frequently associated and have cause and effect relationships When considered as primary factors however the foregoing order of incidence is most probably correct

HEART DISEASE PRODUCED BY INFECTION

Injury to the heart produced by infections is usually overlooked in the train of events occurring during such an episode A patient with pneumococcic pneumonia is concerned about his pneumonia The myocardial injury which is present to a demonstrable extent is ignored because of his anxiety over the pulmonary infection Furthermore the cardiac injury so

3. *Moderately severe acute myocarditis*
   (a) Same as (2)
   (b) Cardiac enlargement to a demonstrable extent by roentgenographic methods
   (c) Signs and symptoms of mild congestive heart failure, usually left ventricular
   (d) Pulsus alternans may be noted
   (e) Accentuated protodiastolic gallop rhythm
4. *Severe acute myocarditis*
   (a) Same as (3) except that all manifestations are more severe. Functional murmurs may be pronounced, the heart is usually extremely large and the signs and symptoms of congestive heart failure exaggerated. The protodiastolic gallop rhythm is more evident
   (b) Decrease in systolic and pulse pressures
   (c) Signs of impending or existing peripheral circulatory collapse

The foregoing discussion from both the clinical and diagnostic points of view is essentially the same for any type of myocarditis whether it is due to diphtheria, postpartal sepsis, Fiedler's myocarditis (isolated acute myocarditis of unknown cause), uremia, direct infection (abscess) of the myocardium or myocardial degeneration as a result of a *systemic infection*. The differences between the types are determined mainly by the systemic or extracardiac manifestations. Toxic states such as ...

The *course* and *prognosis* of toxic myocarditis in the usual patient are excellent. The recovery is usually so complete that no residual is discernible by the methods available to the clinician today. It is quite possible, however, that such injuries may have a definite though subtle influence on the subsequent development of heart failure. In the more severe infections with widespread myocardial damage the patient may progress into congestive heart failure and death from extensive myocarditis. *Acute myocarditis* may be a cause of sudden *death*, especially in young people without any ...

... early proper treatment of infection ...
... the cause ...
... of great ...
... damage is sustained ... infection before any myocardial ...

Although the myocardial injury associated with infections and toxemia is included under the title "myocarditis," the reaction is not usually one of inflammation in the strict sense. The changes represent primarily varying degrees of degeneration, namely, cloudy swelling, hydropic changes, and the like. In many instances the degenerative changes are so mild as to fail to present histologic changes, evidence of myocardial damage being limited to changes in the order of repolarization (*T* wave changes) noted electrocardiographically.

The *clinical manifestations* also vary a great deal. In the average infection, such as pneumonia, sinusitis, tonsillitis or furunculosis, the patient and the doctor are usually unaware of any myocardial damage. The manifestations are usually present, although they may be subtle. The only evidence of injury may be electrocardiographic, that is, depression of the *S-T* segment or depression or inversion of the *T* wave in the standard or precordial leads. At times the patient experiences premature contractions, which in many instances are ventricular in origin. More severe disturbances in mechanism, such as auricular fibrillation and paroxysmal ventricular tachycardia, may be manifested. Disturbances in mechanism are more apt to occur if there is already an underlying disease present. If the myocardial damage is more severe, the cardiac size increases, dilatation may sometimes take place, and precordial discomfort occurs. The pulse rate increases out of proportion to the rise in body temperature, the usual elevation being 8 to 10 beats per 1° F. increase in temperature. The disease may progress into congestive heart failure, left, right, or both, with the manifestations previously described.

If the heart is extremely dilated, there may be many types of *functional murmurs*, especially intense over the mitral area and next in prominence over the aortic area (see section on murmurs). A *protodiastolic gallop* rhythm, a common manifestation, is of considerable diagnostic importance since it indicates myocardial weakness (see discussion of gallop rhythm). The systolic blood pressure and pulse pressure are apt to be low in severe acute myocarditis. Signs of impending or existing *peripheral circulatory collapse* may exist.

The *laboratory data* coincide with what might be expected on the basis of the underlying infection.

The *diagnostic criteria* are:

1. *Mild acute myocarditis*
   (*a*) *Electrocardiographic changes*, such as *S-T* segment and *T* wave changes, changes in the ventricular gradient, *QRS* complex, *P-R* interval, and the like.
2. *Mild to moderately severe acute myocarditis*
   (*a*) Electrocardiographic changes as in (1)
   (*b*) Tachycardia out of proportion to the fever
   (*c*) Cardiac irregularities
   (*d*) Precordial discomfort and palpitation
   (*e*) Protodiastolic gallop rhythm

without any antecedent focus of infection. Previous cardiac disease, acquired valvular deformities and congenital defects, as well as debilitating states, predispose to the disease.

Age. The disease occurs at any age but usually in young adults.

Sex. Males are more often involved than females, the ratio being 3 to 1.

The *pathologic changes* are characteristic. The inflammatory reactions with *thrombi* and fibrin formations vary in size, shape and locations. These *vegetations* or thrombic growths may be fixed to any part of the endocardium. They are usually attached to valvular *leaflets* or *cusps*, *chordae tendineae* and the *endocardial surfaces* of the heart chambers near the valves. These vegetations consist of a mass of colonies of bacteria, fibrin and inflammatory cells. The leaflets, chordae tendineae and endocardium may

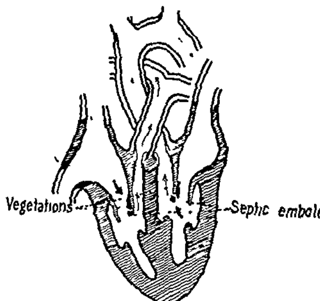


FIG. 131 — ... of the right ... carried throu... of the pulmo... su acute bacterial endocarditis of the left side of the heart emboli escape into the aorta and systemic arteries (dotted arrows) and result in septic embolism of the systemic circulation

become ulcerated. The *septicemia* (bacterem... in ab...

... into blood vessels. The emboli ... arrested in small vessels where they become lodged as a seed or nucleus about which an *abscess* forms. The organs in which the small abscesses form are determined by *the site of the vegetations* (Fig. 131).

and signs of myocardial injury (electrocardiographic tachycardia etc) have disappeared He should be observed closely as he slowly and cautiously regains the ambulatory state and resumes his daily routine Patients frequently die suddenly while attempting to get out of bed for the first time Any signs of myocardial exacerbation indicate a necessity for a return to bed Strenuous exercise or exertion to a state of fatigue should be avoided until the period of postinfectious asthenia has disappeared Cardiac drugs are not indicated except in cases of congestive heart failure or mechanistic disturbances Digitalis is not always efficacious in acute myocarditis It should be remembered that most instances of myocarditis are clinically *reversible*

Endocarditis

Endocarditis is relatively rare as an association or complication of systemic infections however rheumatic fever and lupus erythematosus disseminatus (Libman-Sacks syndrome) may be related to infections in a broad sense The former will be discussed separately At times extra cardiac infections such as pelvic thrombophlebitis sinusitis pyorrhea alveolaris furunculosis and many others may serve as portals of entry for organisms which lodge directly in the endocardium and grow locally to produce *bacterial endocarditis* This is heart disease due to an infection The infections most often *localize* to the *mitral valve* the *aortic* and both *mitral and aortic* in order of frequency The pulmonary and tricuspid valves are rarely infected Congenital defects such as a patent interventricular septum are likely to become sites of bacterial growths

Bacterial Endocarditis —Bacterial endocarditis may be classified by several methods From the clinical point of view it is divided into

1 Acute
2 Subacute

The two differ in clinical severity and etiology as indicated in the following paragraphs

Acute Bacterial Endocarditis —This disease results in recovery or death within two months if untreated with penicillin Since the advent of penicillin the *clinical picture has been altered considerably that is if proper* treatment is administered early

The *etiologic agents* and their incidences are essentially as follows

	Per cent
Streptococcus hemolyticus	40
Staphylococcus aureus	23
Escherichia coli	11
Pneumococcus	9
Gonococcus	4
Meningococcus	4
Other organisms	9

Practically any organism which enters from a focus of infection elsewhere may produce endocarditis and in some instances it may apparently develop

pneumonia or the like The intoxication from the infection may become so pronounced as to result in toxic hepatitis with jaundice as well as other manifestations such as delirium from toxic encephalitis toxic myocarditis or myocardial degeneration

2 The *septic embolic phenomena* should make one suspect this syndrome It is fairly characteristic and of extreme importance in diagnosis (Fig 131) The emboli usually lodge in the tissues of the general circulation such as the skin spleen kidneys [illegible] should be [illegible] tion The [illegible] inflamed [illegible] in the finger tips known as Osler's nodes and small red petechial areas in the skin which blanch on pressure The blanching indicates *dilated engorged blood vessels* in the area immediately surrounding the infected embolus Areas of subcutaneous *hemorrhage* that is purpuric areas *fail to* blanch on pressure since the blood cannot be squeezed away These areas are usually tender They occur anywhere on the skin and for this reason the entire body should be carefully inspected *A single embolism is just as diagnostic as many* Embolism of the eye can be found in the conjunctival area of the sclera and in the fundus of the *eye* on ophthalmoscopic examination Embolism of the kidney manifests itself by pain in the lumbar or renal region tenderness over the *kidneys* on deep palpation definite hematuria and increase in the urinary abnormalities Severe *focal nephritis* may result Embolism of the *spleen* results in splenomegaly detected by *palpation percussion* or roentgenography The spleen is tender and the patient experiences spontaneous *pain over the spleen* Embolism of the [illegible]

ce [illegible]

i [illegible] if the vegetations are on the right side of the heart and the emboli become lodged in the pulmonary structures symptoms and signs of pulmonary disease occur including pain in the chest cough fine moist rales hemoptysis if much pulmonary tissue becomes infarcted and [illegible] evidence of [illegible]

[illegible] he foregoing [illegible] Of course [illegible] *ous cardiac disease* with all of its m[illegible] festations Usually the [illegible] ously damaged v[illegible] valves as well [illegible] damage there is [illegible] a patient to have the clini[illegible] cal syndrome of acute bacterial endocarditis [illegible] so [illegible] se [illegible] he [illegible] qu [illegible] no [illegible] te [illegible] cusps to shrink and become dis[illegible] therefore interfere with closure so that aortic regurgitation with

An embolus may become lodged in the vasa vasorum with abscess formation weakening of the wall of the artery and development of an aneurysm from the high intra arterial pressure This type of aneurysm is known as a *mycotic aneurysm* When an interatrial or interventricular septal defect is present paradoxical embolism (Fig 132) may occur

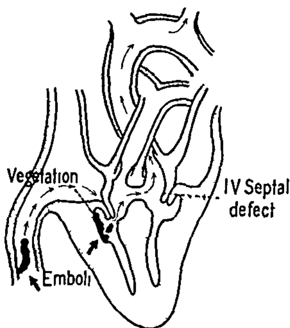


FIG 132 —Illustration showing how a defect in the interventricular septum results in paradoxical embolism Under such circumstances emboli from venous thrombi or from vegetations in the right side of the heart can escape into the systemic or general circulation by passing through the septal defect

The *clinical syndrome* of acute bacterial endocarditis consists primarily of the following interrelated manifestations

1 Severe systemic infection
2 Septic embolism
3 *Cardiac disease or merely a murmur*
4 Positive blood culture

1 The *picture of a severe systemic infection* with high fever is the outstanding initial manifestation This is so true that the clinician unless he is alert is apt to consider in diagnosis some infectious disease other than ... characterized by remittent or ... sweating flushing of ... vomiting lethargy stupor or even coma scanty *urine* of high specific gravity albuminuria slight hematuria cylinduria and leukocytosis with a high neutrophile count This syndrome is not different from that of any other type of pyogenic infection whether it be pelvic thrombophlebitis pneumococcic

pneumonia or the like The intoxication from the infection may become so pronounced as to result in toxic hepatitis with jaundice as well as other manifestations such as delirium from toxic encephalitis toxic myocarditis or myocardial degeneration

2 The *septic embolic phenomena* should make one suspect this syndrome It is fairly characteristic and of extreme importance in diagnosis (Fig 131) The emboli usually lodge in the tissues of the general circulation such as the skin spleen kidneys eyes mesentery brain etc and they should be sought in any patient with a clinical picture of sepsis or infection The emboli manifest themselves in the *skin* by producing tender inflamed nodules in the finger tips known as Osler s nodes and small red petechial areas in the skin which blanch on pressure The blanching indicates *dilated engorged blood vessels* in the area immediately surrounding the infected embolus Areas of subcutaneous *hemorrhage* that is purpuric areas fail to blanch on pressure since the blood cannot be squeezed away These areas are usually tender They occur anywhere on the skin and for this reason the entire body should be carefully inspected *A single embolism is not as diagnostic as many* Embolism of the eye can be found in the conjunctival area of the sclera and in the fundus of the *eye* on ophthalmoscopic examin

in the lumbar or r

definite hematur

focal nephritis may result Embolism of the *spleen* results in splenomegaly detected by palpation percussion or *roentgenography* The spleen is tender and the patient experiences spontaneous pain ov

he heart and the emboli become lodged in the *pulmonary* structures symptoms and signs of pulmonary disease occur including pain in the chest cough fine moist râles hemoptysis if much pulmonary tissue becomes infarcted and roentgenographic evidence of infarction and pneumonitis

3

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aortic val

torted and therefore interfere with closure so that aortic regurgitation with

a murmur develops (consult section on murmur) It is theoretically conceivable that the vegetations will be so small as not to produce murmurs although this rarely occurs in practice The student must be careful to differentiate murmurs due to organic disease of the valves themselves from those due to the toxic myocarditis such as might occur in any severe toxic state

The *electrocardiographic* and *roentgenographic* studies should be included with the usual clinical survey but they in themselves add little in establishing the diagnosis of bacterial endocarditis They are *necessary* studies for the cardiac *inventory* and are important in the complete diagnosis prognosis and therapeutic management but could be omitted as far as the acute lesion of endocarditis itself is concerned

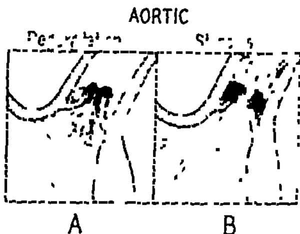


FIG 133 Vegetations on a valve interfere with its closing *and* opening This results in insufficiency with turbulence of flow and a characteristic murmur and stenosis with its accompanying murmur The diagram shows *aortic insufficiency* *A* with a diastolic murmur and aortic stenosis *B* with a systolic murmur Except with extremely small vegetations there is always a murmur of some type if there are vegetations on the valvular leaflets or cusps

4 The *positive blood culture* with the isolation and identification of the offending microorganism completes the clinical picture and establishes the diagnosis as far as is possible with present day facilities

The course of acute bacterial endocarditis in the past was fulminating and 100 per cent fatal Penicillin the *greatest recent therapeutic discovery in cardiology* has changed this considerably Early and adequate treatment has made the course relatively mild and the mortality rate now closely approaches zero With the use of large doses of penicillin the mortality rate will be reduced further

Treatment — The treatment of these patients is simple and of *one* sort only Any other approach today is irrational The measures should include

1 Good nursing care
2 Mental and physical rest in bed
3 Adequate fluids and nourishing foods

4 The organism isolated should be routinely tested for its sensitivity to the antibiotics commonly employed in clinical medicine These organisms are usually sensitive to penicillin and if so it is the drug of choice since it is not likely to be associated with serious toxic side effects The other antibiotics should be employed in proper doses only if indicated either alone or in conjunction with penicillin Remember that antibiotics

lin fortified with crystalline penicillin should be administered parenterally in large doses at least 1 000 000 units intramuscularly every two hours day and night or larger doses

are encounte
patient is ch
two weeks
if any signs

injurious than none at all for they are known to result in the development of penicillin resistant mutant strains of the offending organism thus rendering large doses ineffective when the patient finally obtains proper medical care Remember *if penicillin or other antibiotics fail* there is at present no alternative but death Streptomycin plus penicillin is particularly valuable in certain infections The sulfonamides are of little or no value and can actually create a therapeutic disease (a type of sulfonamide reaction) in an already severely and acutely ill patient

5 Transfusions sedatives laxatives and other supportive measures are

Infective Endocarditis

The subacute type of ... endocarditis is much more common than the acute type It is reported to represent 1 to 2 per cent of all heart disease but it is obviously not this high if all forms of heart disease are included About 7 to 10 per cent of patients with congenital heart disease and about 5 per cent of patients with rheumatic heart disease develop subacute bacterial endocarditis as a complication

The clinical picture of subacute bacterial endocarditis is essentially the same as that of acute bacterial endocarditis except that the course is not as fulminating and the infecting microorganisms are usually different

The etiologic factor in subacute bacterial endocarditis is *Streptococcus viridans* in 90 to 95 per cent of all instances The gonococcus and influenza bacillus account for another 5 to 8 per cent Many other organisms such as *Streptococcus fecalis* may also produce the disease

The *age sex race climate social status* are essentially the same as for acute bacterial endocarditis the latter three factors being of little significance The *predisposing* factors and portals of entry are the same as those described for acute bacterial endocarditis

The *pathologic* changes are essentially the same as for acute bacterial endocarditis but in addition the lesions show evidence of chronicity. The infection may become localized at the openings of an arteriovenous aneurysm, congenital cardiac abnormalities and patent ductus arteriosus. When an artery is infected, *subacute bacterial endarteritis* results.

The *incidence* of valvular involvement is essentially as follows:

	Per cent
(*a*) Mitral and aortic	about 45
(*b*) Mitral	about 30
(*c*) Aortic	about 20
(*d*) Other valves or combinations	about 5

The *clinical manifestations* are about the same as described previously for acute bacterial endocarditis. The syndrome may be divided into the same four main diagnostic manifestations and should be considered in such terms.

Because of the duration of the disease before the advent of penicillin the clinical picture was colored by changes produced by the relatively chronic nature of the disease. Among the important ones are *cachexia, severe secondary anemia, clubbing of the fingers and toes,* and *palmar* and *plantar erythema*. The other clinical manifestations are not different from those of the acute disease except in degree or severity.

In view of the dramatic results obtained with penicillin this *complete* clinical picture has become a clinical curiosity, being present only in those patients erroneously diagnosed or treated or in whom the offending organism is fundamentally resistant to all antibiotics. *The course of the disease* in the past terminated with gradual and almost certain death (97 to 100 per cent). With early diagnosis and proper penicillin or other antibiotic therapy the course should be abrupt and recovery almost 100 per cent. This is a remarkable advance in medicine. The treatment is the same as outlined for acute bacterial endocarditis.

Pericarditis

An inflammation of the pericardium is known as pericarditis. The pericardium may present inflammatory changes in the absence of demonstrable evidence of microorganisms locally. Good examples are pericarditis of uremia and rheumatic fever. Pericarditis, regardless of the cause, occurs in about 1 per cent of routine autopsies.

Pericarditis may be classified according to its pathologic findings, etiologic agent, or clinical picture. The classification presented below is preferred, however.

1. *Infectious*
   (*a*) Septic (*due to specific organism, pneumococcus, tubercle bacillus, etc.*)
   (*b*) Toxic (due to toxin of specific organism, such as rheumatic fever, toxic influence, such as from visceral angitudes, metabolic disturbances, uremia, infarction, etc.)

2. *Physical* (traumatic, etc.)
3. *Neoplastic*
4. *Idiopathic*

This classification may have to be qualified in an individual instance to conform with more generally used clinical terms such as *fibrinous*, *serofibrinous*, *purulent* or *hemmorrhagic* in the case of *acute pericarditis* and *adhesive* (concretio cordis, accretio cordis) and polyserositis in chronic pericarditis.

It will become obvious to the student as he progresses with the discussion that it is often difficult in many instances to differentiate the acute state from the chronic one.

There is *no single pathognomonic sign* that will confirm a diagnosis of chronic pericarditis regardless of the type. In many instances, particularly in the chronic adhesive or constrictive types, the diagnosis is established by eliciting a *syndrome*. Whenever any one of the following is present, the patient has an *inflammation of the pericardium*:

1. Definite pericardial friction rub
2. Paracentesis of the pericardial sac with withdrawal of clear fluid, pus or blood
3. Roentgenographic evidence of air or fluid level in pericardial sac
4. Certain electrocardiographic signs

The clinical syndromes which make a diagnosis possible when such pathognomonic observations have not been made will be presented in the following paragraphs. The clinician should obtain a pathognomonic sign in order to establish an irrefutable diagnosis. When possible, the offending organism should be isolated and identified by bacteriologic methods. This permits an accurate diagnosis to be made, indicates the therapy and establishes the prognosis.

Clinical Aspects of Pericarditis.—The etiologic incidence of pericarditis varies, about 70 per cent being pyogenic, 3 per cent tuberculous and the remaining 27 per cent of various types [illegible] latter type is more common in [illegible] subtropical southern areas. The [illegible] use of antibiotics has been introduced and is modifying the course of primary infections which cause pericarditis. The etiologic incidence should be revised periodically.

Acute pericarditis almost invariably begins with a fibrinous exudate which may persist for a short time at least as *fibrinous* [illegible] fore any [illegible] fibrinous [illegible] bloody [illegible] *purulent* [illegible] or *pneumopericarditis*. It is confusing to classify pericarditis as such, however, because in many, if not all, forms of acute pericarditis exudates change from one form to another so rapidly or so subtly that a diagnosis is wrong or confused by the time the study has been completed. An acute staphylococcic invasion of the pericardium may occur, for example, at 10 A.M. one day as fibrinous pericarditis, become serofibrinous pericarditis thirty minutes later and by 2 P.M., it may be

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blood stream (hematogenous), by direct extension from surrounding mediastinal tissues, by the lymphatics (lymphogenous) or by penetrating wounds.

[illegible]

is, *for practical purposes, always involved in the inflammatory process.* This is particularly important from the electrocardiographic point of view.

A study of the pericardial fluid is important in diagnosis. For this reason it is necessary to determine if the fluid is a transudate (edema type of fluid) or an exudate (fluid of inflammation). The commonly observed characteristics employed in differentiating the two types of fluid are shown in Table 6.

It is obvious from the discussions to follow that the clinical manifestations are variable. They depend upon essentially two main factors:

1. The etiologic agent and underlying disease; and
2. The nature of, and especially the amount of, the pericardial exudate present at that particular time.

1. The underlying disease which serves as the source of the pericarditis, such as pneumococcic pneumonia, rheumatic fever and pulmonary tuberculosis, are responsible for a clinical syndrome of their own. This underlying disease may be so severe and highly developed that the clinician is apt to overlook the complicating pericarditis.

TABLE 6.—DIFFERENCES BETWEEN EXUDATES AND TRANSUDATES

	Transudate	*Exudate*
1. Color	Serous, pale yellow	Fibrinous, purulent, hemorrhagic, chylous or a combination of these
2. Transparency	Clear	Clear or cloudy
3. Specific gravity	Less than 1.018	Greater than 1.018
4. Clotting	*Does not clot*	Clots spontaneously
5. Protein content	Less than 2.5 per cent	Greater than 3 per cent
6. Sugar content	*Same as blood*	Less than blood
7. Cell content	Few (mesothelial and erythrocytes)	Many (type depends upon nature and degree of inflammation)
8. Bacteria	None	Usually present
9. Seromucin (Rivalta test)	Absent	*Present*

Pericarditis is *usually heralded* by *precordial pain* due to the irritation of the surrounding pericardium, pleura and diaphragm. The *pain* may be *continual* or *intermittent*. It is often sharp and lancinating, at times being made worse by *breathing*. The pain is often *referred to the abdomen*. There may be *precordial* and segmental hyperesthesia, and pain on pressure or tenderness is often noted over the *precordium*. There is usually associated *palpitation*. The constitutional signs of *infection* appear if they do *not already* exist, or become worse *if they* were pre-

purulent pericarditis. Such rapid diagnostic differentiation based on the exudate is often impossible. These anatomic and clinical variations are expected in invasive infections of this type. Therefore it is far better and less confusing simply to classify pericarditis according to the etiologic agent involved, *e. g.*, *acute staphylococcic pericarditis*.

Pericardial paracentesis will reveal the nature of the pericardial fluid. This is certainly important in the clinical management, but the character of the fluid has limited diagnostic value. There is a tendency for certain etiologic factors to produce particular types of pericardial exudates.

Fibrinous and *Serofibrinous*

1. *Any infection* of the pericardium
2. Polyserositis (Concato's disease)
3. Uremia
4. Myocardial infarction
5. Foreign bodies
6. Almost any factor which irritates the pericardium

Purulent

1. Pyogenic microorganisms (staphylococcus, pneumococcus, streptococcus, etc.) regardless of their mode of entrance

Hemorrhagic

1. Tuberculosis
2. Neoplasm (primary and secondary)
3. Trauma
4. Fulminating rheumatic fever
5. Rupture of heart or root of aorta (aneurysm)

Hydropericardium (edema fluid in pericardial sac)

1. Congestive heart failure
2. Nephrosis
3. Any edema state, generalized or localized in the cardiac area

Pneumopericardium (air in pericardium)

1. Penetrating wound from
   (*a*) Exterior of body
   (*b*) Lung
   (*c*) Esophagus
2. Erosive lesion from the lung or esophagus

Combinations such as hydropneumopericarditis, serosanguineous exudate, etc., may occur.

Acute pericarditis is found at any *age*, the majority of patients being between ten and forty years. There is a definite *sex* difference in incidence, the male sex being involved three times as frequently as the female.

The *pathologic manifestations* vary with the etiologic agent concerned. For a detailed discussion of these the student is advised to consult textbooks of pathology. The microorganisms reach the pericardium by way of the

blood stream (hematogenous) by direct extension from surrounding mediastinal tissues by the lymphatics (lymphogenous) or by penetrating wounds

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8 Bacteria	None	Usually present
9 Seromucin (Rivalta test)	Absent	Present

Pericarditis is usually heralded by precordial pain due to the irritation of the surrounding pericardium pleura and diaphragm The pain may be continual or *intermittent* It is often sharp and lancinating at times being made worse by breathing The pain is often referred to the abdomen There may be precordial and segmental hyperesthesia and pain on pressure or tenderness is often noted over the precordium There is usually associated palpitation The constitutional signs of infection appear if they do not already exist or become worse if they were pre-

viously present. The severity of the signs of infection is dependent upon the offending agent and constitutional state of the host. The signs of infection are worse if the organism infecting the pericardium is staphylococcus or streptococcus than if it were the tubercle bacillus. These systemic symptoms and signs of infection such as headache, weakness, nausea, vomiting, fever, chills or chilly sensations, malaise, leukocytosis, increased rate of erythrocyte sedimentation, lethargy, delirium, etc., have been discussed previously. The manifestations of infection of the pericardium *per se* may be lost among those already existing. At times when the pericardium becomes infected the new infection causes an exacerbation of the previously existing picture or a recrudescence of symptoms, a cause for suspicion and re-evaluation by the clinician for infection somewhere else which leads him to the pericardium.

Noninfectious disease states such as uremia will present an underlying toxic clinical syndrome which may be characteristic. The pericarditis is an incidental manifestation. The same is true of myocardial infarction or neoplastic disease of the pericardium.

2. The other group of manifestations is dependent upon the nature and quantity of pericardial exudate. For example, the patient with fibrinous pericarditis may present a friction rub (see section on murmurs for description) with few other manifestations of cardiac origin due to the pericarditis *per se*. If the patient has any underlying cardiac diseases such as myocardial insufficiency or valvular disease, these will manifest themselves. Pericarditis with the accumulation of a *large quantity of fluid* results in the syndrome of *cardiac tamponade*.

Pathology and Physiology. The clinical picture produced by cardiac tamponade is not difficult to understand if one is aware of the fundamental disturbance in hemodynamics. The same disturbance and essentially the same clinical picture follows *constrictive pericarditis* (*concretio cordis*). Because of the accumulation of a large quantity of fluid in the pericardial sac, the ventricles are unable to dilate or fill completely during diastole (Fig. 134). The intra-atrial pressure rises, the pressure in the great veins rises, and a generalized and symmetric increase in venous pressure measurements reveals venous hypertension. Even though there is venous hypertension, these patients are often free from edema for some time. Then for some unknown reason the picture of congestive heart failure develops with the cardinal symptoms and signs. Dyspnea is a complaint early in the picture. The rate with which the tamponade syndrome develops depends upon the rate with which pericardial fluid accumulates or constrictive pericarditis develops. The latter occurs at a slow rate, although this may be true for the accumulation of fluid. At times, however, a pericardial effusion may occur quickly. The most rapid tamponade occurs with hemorrhage into the pericardial sac following trauma from the exterior (stab or gun shot wounds) or cardiac or aortic rupture.

In association with impairment of blood flow into the heart there is stasis of blood in the tissues with resultant increase in reduced circulating

hemoglobin This is manifested in those vessels responsible for color of the skin and mucous membranes cyanosis results

With development of severe tamponade *pulsus paradoxus* occurs This is characterized by a *weakening* and *decrease* in volume of the pulse on inspiration In some instances the pulsations of the radial artery may

greatly and perceptibly with inspiration and increasing with expiration The student should consult his textbooks of physiology for the generally accepted explanations The arterial blood pressure behaves directly with

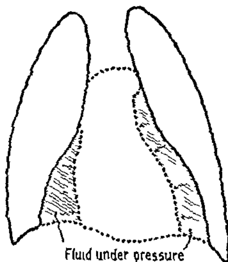


FIG 134 —Diagram illustrating the manner in which extensive pericardial effusion interferes with diastolic filling of the heart and results in cardiac tamponade

the pulse volume Pulsus paradoxus may occur in clinical states other than cardiac tamponade *It is imperative* to check the

... is more *characteristic* than pulsus paradoxus ... with low pulse

Among the many clinical manifestations of pericardial effusion are the signs due to the *mass of fluid* These are obvious and should be remembered

1 Decrease in intensity of the heart sounds or the presence of "distant" heart sound

2 Fullness of the precordial region

3. Decrease in the excursions of the pulsations of cardiac silhouette on fluoroscopic or roentgenokymographic examination
4. ... water bottle (Fig
5.
6. Presence of Ewart's sign that is, evidence of consolidation of that portion of the lungs posterior to the heart (left infrascapular area) This is attributed to compression of the lungs locally by the large mass of fluid There may be other mechanisms involved but none of them has been satisfactorily established

FIG 135 — Diagram of the water bottle shape of the cardiac silhouette in the antero- ... normal heart ... icates the en ... e of the heart ... with air and then making a teleoroentgenogram (see Fig 136)

Diagnosis The diagnosis of an abnormal amount of fluid in the pericardial sac *is established only by withdrawal of the fluid by syringe and needle* Because there is often some question of the source of the fluid withdrawn (it could originate from the pleural space) air should be injected and roentgenographic studies should be made to demonstrate the air and fluid ... (Fig 136) The site and methods of pericardial ... 137 ... me makes one suspect fluid in the pericardium, the collection of the fluid with proper analysis is necessary for diagnosis and determination of the nature of the pericarditis and offending organism

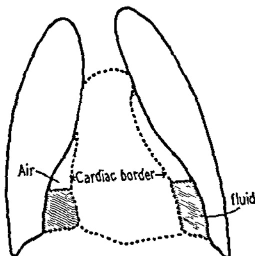


Fig 136 — Illustration of the fluid level and air space in the pericardial sac after partial paracentesis and installation of air This procedure makes it possible to study the size and configuration of the heart and thickness of the pericardium

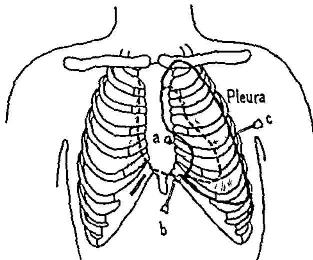


Fig 137 — Three common sites of entrance into the pericardial sac for paracentesis At point *a* the entrance is extrapleural in the fourth or fifth intercostal space just to the left of the sternum At point *b* the entrance is in the costosternal angle and is extraperitoneal and extrapleural At point *c*, the needle penetrates the pleural space and lungs entrance being made in the left posterior axillary or midscapular line The first seems to be the best approach under most circumstances

With the healing of *acute pericarditis*, especially if of long duration and associated with much inflammation, *fibrous tissue develops with scar formation*. This is known as *chronic adhesive pericarditis* since the fibrous tissue produces adhesions between various adjoining structures. Obviously the amount of fibrous tissue and scar formation is variable. If it is diffusely distributed over the epicardial surface, adhesions develop between the epicardium and pericardium. Fibrous tissue scars are prone to contract as they develop and age; therefore the heart finds itself in an *ever-constricting fibrous tissue* jacket which behaves like a tightening jacket of steel (Fig. 138). This jacket interferes with diastolic filling of the ventricles

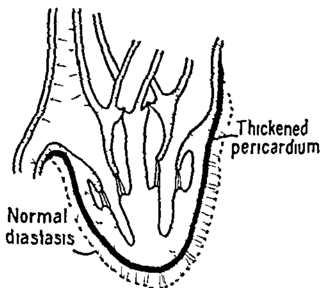


Fig. 138. Diagram of the manner in which the fibrous layer of tissue in chronic adhesive pericarditis encases the heart in an ever-contracting jacket of steel, which results in varying degrees of tamponade.

and results in the *concretio cordis* syndrome of cardiac tamponade. It is essentially the same as the cardiac tamponade due to the accumulation of fluid in the pericardial sac. The main difference obviously is concerned with the cardiac size (Fig. 139); that is, although the *abnormal physiologic clinical pattern may be the same, the heart shadow is large with effusion and small with concretio cordis*. The heart sounds may be impaired in both, and the pulsations of the heart (detected by palpation and roentgenography) are small in both.

At times fibrous adhesions form between the pericardium and the extracardiac mediastinal structures, resulting in what is called *mediastinopericardial adhesions*. These adhesions rarely extend to the thoracic wall so that cardiac movement with each cardiac systole results in a tugging of the intercostal spaces in the vicinity of the heart. This tugging of the intercostal spaces in the posterolateral region of the thorax is known as

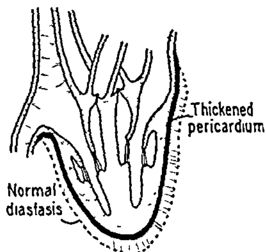


FIG. 139.—Illustration of the manner in which the thick "vest" of fibrous tissue in concretio cordis produces interference with diastolic filling of the ventricles and resultant generalized and symmetric venous hypertension

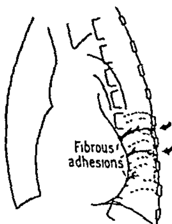


FIG. 140.—The adhesions indicated by the arrows are part of the m

Broadbent's sign (Fig 140) At times the adhesions tend to form around the region of the diaphragm where the inferior vena cava and hepatic veins enter into the mediastinum This results in impairment of venous flow through both veins but more particularly in the hepatic veins because of their angulation and less efficient venous return (Fig 141) Impairment of the flow of blood through the hepatic veins produces venous stasis in the liver with hypertension in the hepatic sinusoids and portal system

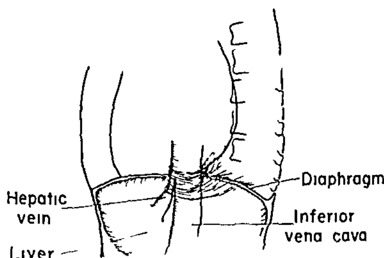


Fig 141 A diagram showing a band of adhesion in chronic mediastinopericarditis which is constricting (1) the inferior vena cava and (2) hepatic veins the latter just before they enter the inferior vena cava This results in an obstruction to the flow of blood from the liver and inferior vena cava Portal hypertension follows with the development of Pick's syndrome (see Fig 142) Obstruction to the hepatic veins is particularly great because of the acute angle at which they enter the inferior vena cava and therefore the ease with which it can be compressed and obstructed and the lack of muscular contractions venous valves and so forth to aid venous return The vena caval obstruction on the other hand is not so great The greater obstruction to the hepatic veins results in ascites and hepatomegaly which are out of proportion to the degree of edema in the lower extremities

This results in a picture like that of hepatic cirrhosis and is known as *mediastinopericarditis pseudocirrhosis of the liver or Pick's disease* (Fig 142) The clinical picture is characterized by enlargement of the liver ascites distended veins of the abdominal wall with local venous hypertension hemorrhoids and other signs of increased parahepatic collateral circulation

s clinical picture in a patient
suspect Pick's disease
a small patch as of scarring
These small white patches
or scars are often referred to as milk spots or soldier's patches

Patients with mediastinopericardial adhesions often have some of the largest hearts encountered in medical practice The dilatation results in many types of functional murmurs which depend on the location and

degree of dilatation One should be careful about the interpretation of these murmurs as signs of organic valvular disease

In a discussion of this nature it is impossible to enter into a detailed discourse of the *management* of acute and chronic pericarditis Only the main principles of therapy, therefore will be summarized and outlined

The *prevention* of pericarditis by adequate early and vigorous treatment of infections and disease states which are known to produce pericarditis should certainly be the main objective of all physicians Once acute

surgically when respo

with removal of small quantities of fluid and its replacement by air, should

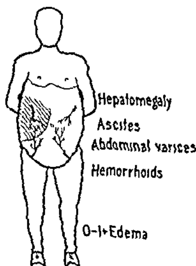


Fig 142 —Diagram summarizing the essential features of Pick's syndrome due to mediastinopericardial adhesions

be employed for cardiac tamponade due to the accumulation of a large volume of fluid Small volumes of fluid should not be removed except for diagnostic purposes *Stab wounds with hemorrhage* must be sutured

being removed only when necessary for reducing tamponade Repeated unnecessary paracenteses will eventually result in *secondary infection* and additional difficulties Medical management *is the therapeutic procedure* of choice Streptomycin and para amino-salicylic acid or the new iso-

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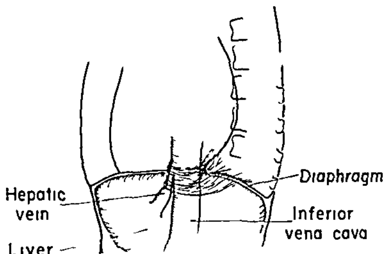


Fig 141 A diagram showing a band of adhesion in chronic mediastinopericarditis which is constricting (1) the inferior vena cava and (2) hepatic veins the latter just before they enter the inferior vena cava This results in an obstruction to the flow of blood from the liver and inferior vena cava Portal hypertension follows with the development of Pick's syndrome (see Fig 142) Obstruction to the hepatic veins is particularly great because of the acute angle at which they enter the inferior vena cava and therefore the ease with which it can be compressed and obstructed and the lack of muscular contractions venous valves and so forth to aid venous return The vena caval obstruction on the other hand is not so great The greater obstruction to the hepatic veins results in ascites and hepatomegaly which are out of proportion to the degree of edema in the lower extremities

This results in a picture like that of hepatic cirrhosis and is known as *mediastinopericarditis pseudocirrhosis of the liver or Pick's disease* (Fig 142) The clinical picture is characterized by enlargement of the liver ascites distended veins of the abdominal wall with local venous hypertension hemorrhoids and other signs of increased parahepatic collateral circulation Edema of the legs and feet is not striking This clinical picture in a patient with heart disease should cause the clinician to suspect Pick's disease

Chronic pericarditis may be mild with only a small patch as of scarring on the epicardium with and without adhesions These small white patches or scars are often referred to as milk spots or soldier's patches

Patients with mediastinopericardial adhesions often have some of the largest hearts encountered in medical practice The dilatation results in many types of functional murmurs which depend on the location and

work in the presence of a lowered oxygen supply Because of the anemia the oxygen-carrying capacity of the blood is reduced The tissues must receive an adequate quantity of oxygen in order to maintain the metabolic processes To meet these requirements in the presence of anemia the blood

the stroke volume and cardiac rate increase the systolic blood pressure rises the pulse pressure increases the rate of blood flow increases, and the cardiac output increases This results in more *cardiac work* in the presence of an inadequate oxygen supply to the myocardium

These physiologic changes do not become manifested until the hemoglobin level falls below 70 per cent of normal Drastic changes do not levels the develops as does the underlying anemia Previous heart disease and debilitating states exaggerate the syndrome and increase the tendency for myocardial damage and failure

The *clinical manifestations* in anemic heart disease are the same as those described previously for acute myocarditis except that there is no tendency towards soreness and pain over the precordium during rest The heart is large *there is palpitation* dyspnea on exertion murmurs of any sort (even diastolic functional ones when the anemia is severe and of long duration) protodiastolic gallop rhythm *congestive* and *anginal* (angina pectoris) heart failure The anginal pain is said to be due to the inadequate oxygen

is the *clinical state responsible for the anemia* if it is secondary anemia

acteristic They include repolarization expressed gments and *T* waves in disturbances in cardiac mechanism may occur These changes though not characteristic are of diagnostic importance in that they *indicate* myocardial damage

For some unknown reason severe g duration are apt to male, namely a large

prevention of anemia The *management* of this disease is essentially as follows

1 *Treatment* —Treatm

(*a*) *Remove*

stasis

respon

nicotinic acid derivatives alone or in combination are the drugs of choice The isonicotinic drugs appear to have considerable promise at this time with the development of other derivatives and more study they may become the drugs of preference In many instances acute pericarditis is reversible if adequate vigorous treatment is instituted early This is particularly true if appropriate antibiotic agents are used

Pancarditis

The term pancarditis is employed to indicate inflammation of the pericardium myocardium and endocardium simultaneously This is a clinical term and has no special significance except for brevity in terminology It is most often caused by acute rheumatic fever although under unusual circumstances acute or subacute *bacterial endocarditis* and lupus erythematosus disseminatus (Libman-Sacks s syndrome) may be associated with pancarditis

HEART DISEASE DUE TO ANEMIA

Anemia is a common clinical entity from the point of view of the world population It is particularly prevalent in the subtropical and tropical regions of the earth where public health measures are poor The fact that there are about 500 000 000 cases of *malaria* every year which produces a severe and chronic type of anemia emphasizes the high incidence of anemia In addition severe anemia is encountered in chronic bacterial infections chronic hemorrhage (from neoplastic states menstrual disorders etc) malnutrition chronic protozoal disease such as intestinal parasites yaws and filariasis chronic degenerative states as nephritis and essential hypertension and in many special types of primary anemias such as pernicious anemia sickle cell anemia and the like Anemia is an important problem even in the most highly developed portions of the earth where public health measures are the best and the heart is one of the organs that is particularly influenced by this state Furthermore the factors producing the anemia are often associated with toxic states which damage the myocardium and further aggravate the patient s condition

The mechanism by which severe anemia injures the heart is not entirely clear At least two main factors are important

1 The anemia itself interferes with oxygen supply to the myocardium at a time when the heart has to perform more work Any tissue which is

the purpose of irrigating the tissues and congestive and or anginal failure supervenes Death follows if adequate early therapy is not instituted

2 A second and important influence on the heart follows the fact that the anemia results in increased demand on the heart so that it performs more

The *management* of this type of heart disease is not difficult as a rule. It is *completely reversible* when therapy is early and adequate since the nutritional disease is reversible. It is likely that subtle changes occur over the course of years from a diet that is only slightly inadequate. The disturbances are so mild that at any one time it is impossible to detect abnormalities but after many years there result demonstrable changes precipitated by some other factor. A good example of this is the present day contention that atherosclerosis is produced in the coronary arteries by an excessive intake of fats and lipids in the diet and an inadequate intake of the lipotropic substances (inositol, choline, methionine). Such may be the case but it remains to be proved. Therefore it is essential to ensure a *proper diet* in all patients whether they consult their physician concerning their heart or for some other reason.

Beriberi Heart Disease

Of the nutritional disturbances, an inadequate intake of thiamine chloride (vitamin B_1)
complex. It
important du [illegible] ... situe ... poverty and inadequate food production and distribution are usually responsible for beriberi throughout the world. Beriberi is not a common disease in the United States, being encountered principally in food cranks, drug addicts, alcoholics, neglected senile people and patients wit[illegible]

[illegible] is unlikely that there has ever been a deficiency of only thiamine in man. Every patient has suffered from a deficiency of many factors of which thiamine inadequacy was the precipitating one. Such deficiencies follow any inadequate intake because of a primary lack of food or because of chronic alcoholism in [illegible]

The *pathologic alterations* in the heart are not ch[illegible]
is diffusely [illegible]
various deg[illegible]
nerves t[illegible]
evidence of [illegible]
[illegible] e myocardium

Before the
[illegible]

(b) Specific measures directed at the anemia itself such as the use of liver extract in pernicious anemia
(c) Transfusions if the anemia is severe Small transfusion (not exceeding 300 cc at a time) should be administered at frequent intervals (every six to twenty four hours) instead of single large transfusions in order to avoid acute left ventricular failure and other serious cardiovascular disturbances

2 The usual management of heart disease such as rest good diet wholesome food treatment of the congestive heart failure Digitalis is not always effective although it has not been shown to produce additional myocardial damage when given in proper doses

The heart disease is completely *reversible* if adequate treatment is instituted early and the anemia is corrected

HEART DISEASE ASSOCIATED WITH NUTRITIONAL DISEASE STATES

Nutritional deficiencies are common throughout the world This is particularly true in the areas where the economic and social status is low In the more highly developed and richer areas these deficiencies are less prevalent but are still common enough to be of importance This is true to some extent in this country In many instances the malnutrition may not be sufficiently great to produce serious heart disease in itself but it must not be forgotten that mild malnutrition will impair myocardial function The malnutrition in most instances is not a deficiency in a single factor but a *chronic dietary deficiency* involving an inadequate supply of most or all dietary substances including vitamins calories essential foodstuffs (carbohydrates proteins fats) minerals etc These chronic dietary deficiencies in a subtle way impair cardiac function to a mild degree Vitamin B_1 (thiamine) deficiency with severe cardiac damage (beriberi) is rare in the United States but relatively common throughout China India the Malayan States and other highly populated areas of the world where the economic level is low *Cardiac damage is seen in pellagra and other deficiency diseases* *Since all of these are multiple deficiencies in man* it is difficult to evaluate the role played by the lack of any single substance

The cardiac damage sustained in malnutritional states like that in anemia is limited to the myocardium The *clinical picture* is essentially the same as that described for heart disease in anemia There is cardiac enlargement tachycardia functional murmurs of many sorts protodiastolic gallop rhythm and finally congestive heart failure In most if not all of the chronic nutritional diseases there is nutritional *anemia as a contributing factor* in the heart disease In fact it is usually impossible to evaluate the relative roles of the two factors for example the malnutrition or *the deficiency* state and the associated anemia

The *management* of this type of heart disease is not difficult as a rule. It is *completely reversible* when therapy is early and adequate since the nutritional disease is reversible. It is likely that subtle changes occur over the course of years from a diet that is only slightly inadequate. The disturbances are so mild that at any one time it is impossible to detect abnormalities, but after many years there result demonstrable changes precipitated by some other factor. A good example of this is the present day contention that atherosclerosis is produced in the coronary arteries by an excessive intake of fats and lipids in the diet and an inadequate intake of the lipotropic substances (inositol, choline, methionine). Such may be the case, but it remains to be proved. Therefore, it is essential to ensure a *proper diet* in all patients whether they consult their physician concerning their heart or for some other reason.

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The *pathologic alterations* in the heart are not characteristic. The heart is diffusely enlarged because of dilatation and hypertrophy, and there are various degrees and stages of degeneration of the myocardium. The nerves to the heart often show evidences of degeneration. There is also evidence of interstitial edema and slight ... myocardium with ...

an ...

...ted illness ... p...olem and 60 per cent at some phase. Of these 99.8 per cent had some cardiovascular manifestations

The *clinical manifestations* of beriberi heart disease are altered to a variable degree by the underlying manifestations of *nutritional deficiency*. The ... must ... of th... manifestations of beriberi are

- Peripheral neuritis
- Psychosis and other mental alterations
- Glossitis
- Constipation or diarrhea
- Dermatitis
- Anemia
- Hypoproteinemia
- Optic neuritis
- Dysphagia
- Hoarseness
- Aphonia
- Koilonychia
- Purpura
- Weakness and fatiguability, etc.

The cardiovascular manifestations are

Symptoms

- Tachycardia and palpitation
- Fatiguability and weakness
- Dyspnea on exertion
- Cough
- Edema (cardiac type)
- Orthopnea
- Paroxysmal dyspnea

Signs

- Tachycardia
- Embryocardia
- Systolic murmurs
- Diastolic murmurs when the disease is severe
- Prominent cardiac pulsations
- Pulmonary rales
- Gallop rhythm
- Venous hypertension
- Enlarged heart
- Warm extremities
- Pistol-shot sounds in the large arteries
- Cyanosis
- Syncope
- High pulse pressure
- Circulatory collapse, etc.

These manifestations are obviously those of myocarditis, congestive heart failure, high pulse pressure and peripheral circulatory failure which ha... ...racteristic. They are of the ty... ...al degeneration and include lowering and inversion of the *T* wave, prolonged *Q-T* interval, low voltage of the *QRS* complex, sinus tachycardia, disturbances in cardiac mechanism, etc.

The changes in hemodynamics other than those due to congestive heart failure are essentially the same as those described for *heart disease in*

anemia In fact the associated anemia may be largely responsible for the changes Some of these are

1 Normal or increased volume and linear velocity of blood flow and increased cardiac output
2 Low arteriovenous oxygen difference
3 Full and bounding pulse, flushed skin, high pulse pressure and general signs of arteriolar dilatation
4 Signs of purely right ventricular congestive heart failure
5 Signs of peripheral circulatory collapse and shock

There are certain *chemical changes* in the blood which are related to the vitamin deficiency itself and not necessarily to the cardiac disease These include

1 Decreased plasma proteins and specific gravity
2 Normal or moderate increase in nonprotein nitrogen
3 Moderate elevation of fasting blood sugar
4 Increased bisulphite binding substances
5 Tendency to ketosis and glycosuria

The *diagnosis* is usually not difficult It may be confused with obscure types of myocarditis and vasomotor disturbances

Treatment—The treatment is obvious *Thiamine chloride* should be administered in daily doses of 50 to 120 mg *orally* If necessary, 50 mg should be given *intravenously* *Bed rest* should be obligatory The diet must be adequate and especially high in vitamins and calories Transfusions sedatives and other supportive measures must be employed The usual measures employed for congestive heart failure are indicated only when failure is present *Digitalis* may be tried but does not seem to be especially *effective* in beriberi heart disease It is not known if digitalis will further damage the myocardium

CARDIAC DYSFUNCTION PRODUCED BY PSYCHONEUROSES

There may be a great deal of discussion as to whether or not the disturbances in cardiac function produced by *psychoneurosis* or *cardiac neurosis* should be considered as *heart disease* Any conclusion reached by such discussion would remain a matter of opinion From the practical clinical standpoint the person who is concerned about his heart or who suffers from heart trouble because of a psychoneurosis is a medical social, and economic problem He is unhappy and is not as efficient as the man who is free from such difficulties It is true that in most instances his heart can perform a normal amount of work but it is also true that the individual *is unable or unwilling to* permit his heart to work fully In some instances the patient becomes an invalid and is a slave to his heart beat Finally these people forever confront their physician with their complaints shop for medical assistance and become the victim of the charlatan Their home *life and* business associations are often seriously disturbed Furthermore *the cardiac neuroses* are extremely common and constitute an important clinical problem which is often badly managed

They are *reversible* in many instances. The physician has much to offer these people. Their cardiac status is as amenable to treatment as the *underlying psychoneurosis. On the basis of these facts the cardiac neuroses* are considered among the most common types of cardiac dysfunction even though they are not organic in nature.

The *cardiac neuroses to be discussed* are:

1. Simple paroxysmal tachycardia
2. Neurocirculatory asthenia
3. Gowers's syndrome (cardiac component of the vasovagal syndrome)

The relationship of emotion and cardiovascular phenomena is well known. Tachycardia, sighing respiration, giddiness, syncopal episode, palpitation, precordial and substernal pain are among the many experiences of a patient with anxiety states. It is obvious that any classification of the cardiac neuroses is inadequate. Since the foregoing three types of cardiac neuroses occur in variable degrees, almost all patients with cardiac neurosis can be placed in *one of these categories. Many people who suffer from* psychoneuroses have such a mild cardiac neurosis that no attempt is made to render a cardiac diagnosis. Such a mild form of cardiac neurosis has potentialities, however, of becoming fully developed into a serious problem.

Simple Tachycardia. A reasonable number of people under the influence of anxiety states have episodes of tachycardia which frighten them considerably. The normal person may experience these episodes without too much *alarm and without development of an exaggerated* state of anxiety. The individual who already suffers from anxiety neurosis, however, has a fertile psychic background for engendering a state of great fear of heart disease and sudden death. He then lives in dreaded anticipation of a cardiac death. Newspaper articles and reports of sudden cardiac deaths, the recollection of relatives and friends who had cardiac disease and tachycardia and died of heart disease add fuel to the fire of the cardiac neurotic. He becomes so conscious of his heart that he is forever checking it by palpating his radial artery and precordium, anticipating trouble. A premature beat or sinus tachycardia in response to an emotional experience, a normal cardiac reaction, produces considerable concern and fear about his heart. *This fear results in more tachycardia and more fear.* Soon a typical fear syndrome follows with pallor, sweating, weakness and near collapse. This is often superimposed on a syndrome of hyperventilation. The patient soon finds himself helpless and dependent on his immediate attendants for assistance and psychic support. He loses confidence in his health, his heart and himself and expects death at any moment. A capable and understanding doctor can step in and take control only if he can quickly appreciate the situation, administer the proper advice and suggest therapy and psychiatric measures.

The attacks of tachycardia are usually (1) paroxysms of sinus tachycardia, (2) paroxysmal tachycardia, (3) auricular flutter or (4) auricular fibrillation. *Caffeine beverages and tobacco* are likely to increase the frequency, severity and duration of the attacks of tachycardia.

These episodes of tachycardia occur between the *ages* of twenty-one and seventy-six years the average age being fifty They are found in both *sexes* and in all *races* There is a tendency for the patients to suffer with stammering colonic dysfunction, pyrosis, hyperventilation, migraine, aero-

Heart, Effort Syndrome, Irritable

...e best of the foregoing terms, is a symptom complex which is characterized by fatigue, exhaustion, palpitation dyspnea (often only sighing respiration), vertigo, sweating, weakness, precordial pain or discomfort, headache, tachycardia and anxiety neurosis The syndrome is often precipitated by severe infections emotional excitement, physical or psychic strain

This syndrome always occurs in people who are hypersensitive and suffer with a psychoneurosis (usually anxiety neurosis) It is most likely to manifest itself or become fully *developed* during periods of psychic stress ... difficulties or any period ... In fact, Da Costa first

... said to be great being present in about 10 per cent of civilians and reaching much higher levels during war time The *age* varies between twenty and forty years *The sex* incidence is unequal about 60 per *cent of the cases* ...
important in that cer...
incidence *Environ*...
petitive business and enterprise marital financial and other constant worries bring on the syndrome Tobacco alcohol caffeine beverages infections etc may be precipitating factors

There are no known *pathologic* changes

The clinical *manifestations* are variable *Palpitation* ... al pain or ach...
... is a tend-
... Fatigue
... nervousness giddiness peripheral circulatory collapse (pallor and cold clammy hands) sighing respiration and instability of the blood pressure and heart rate are among the many complaints

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ps... difficulties ... an escape mechanism from their ... Such people usually refuse to ad...
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and n...

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Neurocirculatory Asthenia (Soldier's Heart, Effort Syndrome, Irritable Heart) —Neurocirculatory asthenia, the best of the foregoing terms is a symptom complex which is characterized by fatigue exhaustion, palpitation dyspnea (often only sighing respiration) vertigo sweating weakness precordial pain or discomfort headache tachycardia and anxiety neurosis The syndrome is often precipitated by severe infections emotional excitement physical or psychic strain

This syndrome always occurs in people who are hypersensitive and suffer with a psychoneurosis (usually anxiety neurosis) It is most likely to manifest itself or become fully developed during periods of psychic stress such as during wars domestic or financial difficulties or any period requiring an important decision for the individual In fact, Da Costa first described the syndrome during the Civil War

Incidence —The incidence is said to be great being present in about 10 per cent of civilians and reaching much higher levels during war time The *age* varies between twenty and forty years The *sex* incidence is unequal about 60 per cent of the ca...

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There are no known *pathologic* changes

The *clinical manifestations* are variable *Palpitation precordial pain* or *aching* and *dyspnea* are the most common ... is a tendency for exhaustion to ... Fatigue faintness syncope ver... giddiness peripheral circulatory ... (clammy hands) sighing respiration and instability of the blood pressure ...

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o ... ise are often welcomed as an escape mechanism from their psychic difficulties Such people usually refuse to admit the ... psychic illness ... and inadequ...

evidence in favor of organic disease and against mental disease. For this reason, some patients cherish their cardiovascular disturbances, thereby increasing difficulties in therapy.

The *physical and laboratory examinations* fail to show any abnormalities. Results of *electrocardiographic*, *roentgenographic* and *metabolic* studies are normal. There is a high incidence of a *sharply localized area of tenderness* over a rib or *portion of the sternum in the precordial region*. Pressure over this area elicits pain which is identical with the spontaneous pain previously experienced by the patient. The sign is of *considerable* diagnostic value not only in support of the
and anxiety neurosis but also in exc
The *dyspnea* is more subjective th
respiration a pathognomonic sign of psychoneurosis. This is as incapacitating to the patient as the dyspnea encountered in congestive heart failure.

The four cardinal symptoms of neurocirculatory asthenia are:

1. Palpitation
2. Respiratory discomfort
3. Precordial pains or aches
4. Exhaustion

The *palpitation* (consciousness of the heart beat) is so highly developed at times that these patients are almost constantly cognizant of their heart beating. Such a person will observe his heart beat for fear it will suddenly stop and cause his death. He is unable to sleep on his left side; when he is lying in bed the squeaking sounds that the cardiac pulsations produce in the bedsprings frighten him and excite attention. He notices with concern his abdominal wall vibrating, his ears throbbing, and his precordium and overlying clothes pulsating with each cardiac ejection. These pulsatile phenomena alone may be responsible for almost a state of panic from fear.

The *dyspnea* and precordial pain or ache have been discussed earlier in this monograph.

The *exhaustion* and fatigue from which these patients suffer are of considerable importance to them. Because of it, they have little interest in work or play. Any participation in mental or physical activity is brought about only with effort. They must force themselves to work, and when work is undertaken, extreme fatigue occurs quickly.

There are, of course, varying degrees of any disease, including neurocirculatory asthenia. The well developed syndrome is not difficult to recognize. On the other hand, only the astute and patient physician who studies his patients carefully and evaluates the history well will succeed in recognizing mild and early forms of the syndrome. Early diagnosis is essential for successful therapy.

Vasovagal Attacks (Gower's Syndrome). — The *incidence* and background for Gower's syndrome are essentially the same as that described for neurocirculatory asthenia, but this syndrome *is not as frequently* encountered as the latter. Most of these attacks are ignored or simply diagnosed as "anxiety attacks," "heart attacks," "vertigo," or some such vague entity.

The syndrome has a definite mechanism which can become highly developed and incapacitate the patient. It therefore deserves serious consideration. Although it is not as common as neurocirculatory asthenia, it may become more severe. There is no reliable *pathologic pattern*.

The *clinical picture* is fairly characteristic. In brief, the symptom complex consists of:

1. Intense discomfort and physical prostration persisting for hours or days.
2. The appearance of being *in extremis*.
3. Similarity *to coronary occlusion and angina pectoris*.
4. A syndrome from which the term vasovagal originates, consisting of:
   (a) Facial pallor.
   (b) Pale, cold and sweaty extremities.
   (c) Sublingual cyanosis.
   (d) Weakness.
   (e) "Glassy" or "glarey" appearance to the eye.
5. Contracted and almost pulseless radial arteries.
6. Sense of thoracic constriction, discomfort, and actual pain in the substernal and precordial regions and left arm and neck.
7. Severe malaise.
8. Helpless immobility.
9. Leaden feeling in the arms.
10. Tingling in the arms and trunk.
11. Tetany (rarely).
12. Coldness and chills.
13. Loss of consciousness (rarely).
14. Objective observations such as:
    (a) *Pallor*.
    (b) *Coldness* of the body surface.
    (c) Extreme retardation or acceleration of the *pulse*.
    (d) Shallow or rapid *breathing*, which may produce the *hyper-* [illegible]
    (e) [illegible]

Because th[illegible] frequently be[illegible] ered the syn[illegible] *be the result of a violent vagal* stimulation with severe vascular reactions.

The background of anxiety neurosis, mental conflicts, infections, tobacco, alcohol, chronic illness, poor environmental conditions [illegible] e sent [illegible] vari[illegible] tired[illegible]

Management of the Cardiac Neuroses

The management of the cardiac neuroses is usually not difficult if the physician is aware of the psychoneurotic background and environmental difficulties underlying it. The treatment is essentially the same for all

evidence in favor of organic disease and against mental disease For this reason some patients cherish their cardiovascular disturbances thereby increasing difficulties in therapy

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The *dyspnea* is more subjective than objective It is usually *sighing respiration* a pathognomonic sign of psychoneurosis This is as incapacitating to the patient as the dyspnea encountered in congestive heart failure

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When it is realized that there are at any one time 15 million or more people in the United States alone who suffer from a form of cardiac neurosis it is easy to realize the importance of the syndromes clinically. Although these people are definitely sick the disease is clinically reversible with adequate and careful management.

HEART DISEASE PRODUCED BY THE AGING PROCESS (SO CALLED ARTERIOSCLEROTIC HEART DISEASE)

This and the next five etiologic types of heart disease are usually considered the "six common types" of heart disease. They are the ones which command most attention by clinicians as well as in the textbooks of medi

consider the arteriosclerotic changes in the coronary arteries alone responsible for the cardiac disease. This attitude exists because it is relatively simple to demonstrate vascular changes histologically and to theorize mechanistic phenomena to explain the disease. Surely it is possible to exceed existing data but many subtle physical and chemical phenomena must occur in the cardiac parenchyma independently of the circulatory changes resulting from the coronary sclerosis. Although the likelihood of *other phenomena must be kept in mind* it is necessary for want of data that this discussion be limited to the incomplete and inadequate conventional concepts of impaired coronary circulation. The more general term *Heart Disease of the Aging Process* is employed here to avoid too definite a commitment to the idea that the cardiac disease is the result of arteriosclerosis. The exceedingly important process of arteriosclerosis is obviously included.

The *etiology* of the aging process is unknown. It is inevitable, occurring relatively early in some people and rather late in others. There seem to be genetic factors concerned with the rate, degree and quality of aging. The environment in regard to such factors as climate, infections, diet, psychic and physical trauma, rest and the like plays an important role. This aging process produces demonstrable histologic changes within the parenchyma and the coronary vessels, usually as atherosclerosis ... in the ...

... process permits compensating and collateral adjustments to take place, whereas rapid processes may occur so quickly as to result in massive death of the ...

The *age* of ...
forty year...
fact it is ...
changes in the heart
increases with age

such cases, and for practical clinical purposes they are *reversible*. Many of these patients who have been slaves to their illness become extremely grateful, for they generally receive little serious consideration from physicians and are usually poorly managed. It should be realized that nothing can be offered them by drugs alone or a pat on the back. In many *instances the syndrome follows or is aggravated by a busy physician's* casual remark about a leaky valve, a weak heart, or a murmur. Such terms must never be employed carelessly in their presence. Most of these patients are intelligent and *all of them are sick*. They are just as sick from any point of view (except life and death) as the patient with coronary occlusion, and they should be treated with this in mind.

There are essentially two approaches to the treatment of the cardiac neuroses:

1. The management of the psychoneurosis and environmental difficulties.
2. A thorough medical and cardiovascular survey to detect and rectify any physical disturbances. It is important to reassure the patient that you are certain of his health because *you have really investigated it*. He knows that a glance at the throat or a thump on the chest does not constitute a thorough investigation or grounds for any dogmatic statement that "your illness is not organic but functional." The physician must first convince his patient that he has accumulated sufficient reliable data to render a definite statement concerning his health. Such an examination requires time, effort and usually *expenditure of much money, but this is necessary*.

The *management of the psychoneurosis*, usually an anxiety neurosis, is absolutely necessary for success in treatment. This cannot be accomplished by drugs or even aided much by them but rather requires many hours of careful questioning and discussion. The average physician should be qualified to handle the average patient. Every good physician is a good psychiatrist. A detailed discussion of a proper psychologic approach to such a problem has no place here. In most instances the management is obvious but requires adequate personability and intelligence on the part of the physician. It is a mistake to begin to unravel the patient's conflicts and to reveal them to him without following through with a satisfactory solution. As long as the problems remain, anxiety will remain. As soon as his problems have been solved they cease to exist as problems in his mind *and therefore he cannot be anxious about them*. Most patients are dramatically relieved of their cardiac neurosis when the anxiety neurosis is relieved. It is well to reiterate that drugs should rarely be used. Occasionally sedatives may be prescribed or a drug may be employed for the tachycardia if it becomes annoying, frequent or severe. Foci of infections should obviously be removed. There should be no indiscriminate removal of teeth and tonsils. Mental and physical rest, occupational therapy, frequent vacations, good diet and hygiene should be emphasized. The patient should be given a sense of self-confidence, even overconfidence, if necessary.

A detailed description of all possible isolated lesions that might follow the aging process and coronary artery sclerosis would not be possible here. It is obvious that the pathologic changes and resultant disturbance in physiology would vary with the degree and location of the disease process. For example, if the superior perforating branch of the posterior descending coronary artery becomes moderately obstructed, the atrioventricular node would be deprived of some of its blood supply and its function would be impaired. This would result probably in slight prolongation of the P-R

TABLE 7.—INCIDENCE OF CORONARY ARTERY DISEASE

	Per cent
Age incidence of clinical coronary heart disease	
Below 40 years of age	less than 0.5
40 to 50 years of age	6.0
50 to 60 years of age	23.0
60 to 70 years of age	44.0
Over 70 years of age	27.0
Age incidence of angina pectoris	
30 years of age and under	1
31 to 40 years of age	3
41 to 50 years of age	21
51 to 60 years of age	41
61 to 70 years of age	26
71 to 80 years of age	7
Over 80 years of age	0
Age incidence of myocardial infarction due to coronary thrombosis	
Below 30 years of age	1
30 to 40 years of age	4
40 to 50 years of age	17
50 to 60 years of age	36
60 to 70 years of age	31
70 to 80 years of age	10
Over 80 years of age	1

TABLE 8.—LOCATION OF INFARCTS IN THE LEFT VENTRICLE

	Per cent
Anterior portion and apex	44
Median portion	13
Posterior basal portion	38
Subendocardial (diffuse)	5

TABLE 9.—INCIDENCE OF OCCLUSION OF THE CORONARY ARTERIES

	Per cent
Anterior descending branch of left coronary	53
Circumflex branch of left coronary	21
Right coronary	26

coronary heart disease are beyond sixty years of age about 75 per cent of patients with angina pectoris are beyond fifty years of age and about 80 per cent of those with myocardial infarction are beyond fifty years of age (Table 7) However it must be remembered that patients in the third and fourth decade of life may also present these types of heart disease

The *incidence* is about the same for *both sexes* Men tend to have more evidence of clinical coronary disease than women the ratio being about 2 or 3 to 1 Robust masculine and plethoric persons are more prone to coronary disease

Race temperament social activity social status and *economic status* have little influence on the incidence The professional and business groups are said to be prone to coronary disease

Alcohol coffee and *poor diet* appear to increase the rate of aging but this has not been proved to be significant in man At the present time a diet high in fats is said to predispose to atherosclerosis Cholesterol is considered to increase this tendency However this has never been established for man Furthermore man can produce fat from carbohydrates anyway

Pathologic changes that occur in the aging process will not be discussed here nor even atherosclerosis either in general or in the heart and coronaries in particular Changes occur within the intima and the muscle fibers of the coronary arteries and the surrounding structures The atherosclerotic changes in these arteries result in an insufficient supply of blood to the myocardium This in turn produces degenerative changes in the myocardium manifested by hypertrophy cloudy swelling and hydropic degeneration fragmentation of the fibers and accumulation of fibrous tissue in the interstitial spaces All vessels show signs of degeneration The work of the heart continues however even though its blood supply is being reduced insidiously This results in relative insufficiency as the lumina of the coronary trunks and their branches become slowly reduced in size However some collateral circulation develops through the Thebesian and arteriolumninal vessels At times a coronary vessel may be rather quickly and completely occluded by thrombosis before collateral circulation develops This results in the death of a mass of muscle or *infarction* of the myocardium with the characteristic pathologic picture of infarction The incidence of involvement of various portions of the left ventricle is shown in Table 8

The reasons or mechanisms for the high incidence of thrombosis of the coronary arteries are unknown This is also true for the frequency of distribution among the different coronary arteries and their branches (Table 9) With infarction and weakening this segment of dead muscle fails to contract and is ballooned out by the high intraventricular pressure resulting in the formation of an *aneurysm* of the ventricle This weakened area is friable and made especially weak by the proteolytic and other enzymes released locally by the dead cells and inflammatory cells The area sometimes is so weak that a *rupture of the heart* and hemorrhage into the pericardial sac with tamponade occurs

It is well to point out at this time that any disease associated with impairment of coronary blood flow such as *thromboangiitis obliterans scleroderma* or *periarteritis nodosa* produces clinical changes similar to those described later for arteriosclerosis of these vessels

The *clinical manifestations* vary considerably They are usually those of

1 Impaired coronary circulation
2 Myocardial insufficiency (discussed at end of Chapter)

Impaired Coronary Circulation

Impairment of coronary artery blood flow produces (a) myocardial infarction and/or (b) angina pectoris

(a) **Myocardial Infarction** — Myocardial infarction is characterized by

1 *Sudden severe pain* in the substernal precordial and epigastric regions The pain is squeezing and vise like in character and extremely severe or almost unbearable The pain is referred to the left shoulder, left side of the neck left arm along the ulnar side and at times is referred to any part of the upper and even the lower abdomen and extremities when it is severe It is not relieved by rest or nitrites and only partially by morphine It lasts over fifteen minutes and usually several hours gradually lessening in intensity *In only about 60 per cent of the instances is the pain typical in location duration intensity or quality* It may even be absent

2 ... elop rapidly This is ... pallor cold sweatin

3 ... or from left ventricular congestive failure

4 *Palpitation* is a common manifestation which is annoying to the patient It may be aggravated by many types of cardiac irregularities of which premature beats auricular fibrillation ...

6 *Fever leukocytosis* and an increase in the rate of *erythrocyte sedimentation* occur within twelve to twenty fo...

... mononuclear cells and gradually returns to normal in about forty-eight to ninety six hours or more The erythrocyte sedimentation rate is usually the last of these to return to normal The magnitude of these reactions is essentially directly proportional to the size of the infarct

7 A *friction rub of the pericardium* may occur when the infarction is fairly large and enough fibrinous epicarditis is present This may be

interval. If this vessel were completely occluded the AV node would become infarcted and complete heart block would result (Fig. 143). Essentially the same state of affairs could produce bundle branch block, S-A nodal block, etc. For this reason the aging process may manifest itself in *many ways* too numerous to mention.

The aging process may result in deposition of calcium and fibrous tissue in valves and in the regions of the attachment of valves resulting in *calcular heart disease*. The valves most often involved are the aortic and mitral; rarely are the other two affected. It is believed by many observers that





Fig. 143.—Diagram illustrating the mechanism by which arteriosclerotic occlusion of the superior perforating branch of the posterior coronary artery *A* produces interference with the nutrition of the *AV* node *B* with resultant complete *AV* block (lower part of figure). The small black blocks indicate auricular activity; the lower ones are sounds of the black auricular.

calcification occurs only in valves previously diseased, as by rheumatic fever. Calcification of the aortic and mitral valves results in aortic stenosis and insufficiency, the stenosis being most significant in the aortic valve and the insufficiency most important in the mitral. These valvular defects interfere with hemodynamics to an extent sufficient to increase the work of the heart. When there is calcification of the valves it is almost certain that there are atheromatous changes in the coronary arteries as well. This only aggravates the situation, for the valvular disease increases the cardiac work at the same time that the coronary disease is depriving the myocardium of adequate blood supply.

Therefore, the *aging process* injures the heart by essentially *three primary* pathologic processes:

1. Atherosclerotic changes within the coronary arteries.
2. Valvular disease.
3. Aging of the parenchyma *per se* of the heart.

These factors may exist separately or in various obvious combinations. The second occurs least frequently.

5 Atropine sulfate (gr 1/150 or 0 3 mg) may be used also but it is certainly not indicated routinely

6 Oxygen should be used as early as possible, preferably with the patient in an oxygen chamber or tent and at concentrations of 55 per cent The oxygen and cool tent are exceedingly important agents If the patient does not object it should be used even if there is no evidence of cyanosis Employ the best method available

7 In the presence of congestive heart failure *digitalis* should be used as well as *salt restriction* and diuretics The latter two may suffice and may be more important Salt (sodium) restriction should be employed routinely with all infarctions

8 *Quinidine* (gr 6 or 0 4 Gm every two to four hours) is used for ... or frequent ... is used ... quinidine fails It must be employed cautiously especially in irregularities of ventricular origin because of the tendency toward ventricular fibrillation and death

9 *Liquid or soft diet* (for example, baby foods) may be given for the first few days if the patient desires food The amount may be increased and vitamins may be added as necessary *Carbohydrates such as candy* should be allowed and even ordered therapeutically

10 *Caffeine beverages tobacco or alcohol* should be avoided

11 The patient should be instructed to *move his legs* around slowly in bed to *avoid thrombophlebitis*

12 A *low salt diet* should be *routine* until the myocardial reserve has been adequately estimated

13 *General hygienic measures* should be *instituted* The stool should be kept soft with *laxatives to avoid* straining and associated disturbances in hemodynamics during bowel movement Purgatives should *not be used*

14 *Drugs and stimulants* should be avoided They do more harm than good The patient's body *not only* has to overcome his disease but the various drugs *administered* Intravenous medication and fluids should *not be used unless* they are absolutely necessary

(*b*) **Angina Pectoris** — Angina pectoris is frequently associated with aging especially with coronary artery *sclerosis* It is often associated with *other* types of cardiac disease such as that due to ...

... incidence varies a great deal It is found in all races sexes and regions of the earth It is more common in *men* than *women* and less common in *Negroes* It is usually asso... per cent) with organic ... process It increases ... people working under

transient, and therefore the patient should be watched closely in order that it not be overlooked

8 There are *miscellaneous manifestations* present, such as faintness, syncope, nausea, vomiting, convulsions, mental disturbances of varying degree and even coma *Adams-Stokes* syndrome (vertigo, faintness, syncope, convulsions and bradycardia), due to complete AV block, may occur if there is a posteroseptal infarct

9 *Congestive heart failure* with its various manifestations may develop at some time during the immediate or subsequent course of the infarction

10 *Physical examination* will reveal these manifestations plus cardiac enlargement in many instances and murmurs that might have been present previously A soft blowing systolic mitral murmur is likely to develop This is referred to as a functional murmur There is nothing particularly characteristic of infarction on examination except possibly the friction rub Any previous disease states, such as might be associated with senility and arteriosclerosis, are noted if present

11 *Roentgenographic* examination usually shows nothing characteristic of infarction except a *ventricular aneurysm*, if present, or an area of *muscular inactivity* on a roentgenokymogram or fluoroscopic examination when this is performed There may be cardiac enlargement due to a previous disease or because of the infarct

The *course* of myocardial infarction is variable It depends upon the size, location, complications of the infarct and general health of the patient The patient may live only a few hours or days About 20 per cent die during the first month, whereas 25 per cent survive ten or more years With prompt and adequate treatment the prognosis is good for the first infarction The incidence of death increases rapidly with the number of infarctions Such *complicating states* as impaired renal function old age, shock, congestive heart failure high fever and leukocytosis, acute left ventricular failure, gallop rhythm heart block, pulsus alternans, paroxysmal tachycardia, pneumonia or similar systemic diseases make the prognosis worse

Among the *complications* resulting from infarction are cardiac aneurysm cardiac *rupture*, *disturbance in mechanism* *congestive heart failure*, thrombo-embolic phenomena and ventricular fibrillation with death

The *treatment* for uncomplicated myocardial infarction may be outlined as follows

1 *Absolute rest* in bed with complete elimination of psychic disturbances for at least six weeks Bathroom privileges are *not* allowed
2 Comfortable and pleasant *surroundings* with adequate and careful *nursing*
3 *Morphine* sulfate in adequate doses only (gr ¼ or 15 mg) repeated as often as necessary for the pain
4 *Visitors* or *disturbing guests* should not be permitted for the first two or three weeks

A number of patients die suddenly during an attack of pain and many develop coronary occlusion In some instances the angina pectoris follows an attack of myocardial infarction presumably arising from adjacent vessels The converse is also true in that chronic angina pectoris may be cured by infarction of the involved area The degree of invalidism varies considerably Some patients have such frequent and severe episodes that they are almost completely incapacitated Of course a clinical cure is also possible and occurs not infrequently

Such *complications* as auricular flutter and fibrillation paroxysmal tachycardia other irregularities of the cardiac mechanism *coronary occlusion* and congestive heart failure may occur

The *treatment* of angina pectoris consists in the following measures

1 *Mental and physical* rest is the most important requirement the nature of the rest varying with the circumstances Absolute rest in bed should be prescribed if the episodes are frequent and severe If they are mild infrequent and associated with hard work or business pressure a vacation in a different environment is indicated Long periods of sleep rest in bed after meals and the like should be advised *Physical and mental* activity should never extend to the point of producing pain *It is essential that any anxiety* state be properly managed this can usually be done by the average clinician a psychiatrist not being necessary

2 *Infections problems of diet sleep* and the like should be given serious consideration as described previously for *coronary occlusion* *Tobacco caffeine beverages* and *alcohol* should be forbidden

Drugs except the nitrites are of little use Nitroglycer...

... and is just as satisfactory *Sedatives* (phenobarbital in doses adequate to obtain the desired effects) should be employed only when absolutely necessary *Laxatives* may be used if necessary *Digitalis* and *quinidine* should be used for auricular flutter or fibrillation and paroxysmal tachycardia as previously indicated

The role of *surgical therapy* has not yet been evaluated

HYPERTENSIVE HEART DISEASE

Diastolic hypertension produces heart ...

... out the myocardium
an extra load but also ...
... and damaged myocardium

extreme psychic tension or suffering with anxiety neuroses. The professional and business groups are especially susceptible to angina pectoris.

The mechanism is unknown. It is thought to be due to temporary coronary artery spasm with local impairment of the blood supply to the myocardium. It is known, however, that circumstances which produce arterial spasm elsewhere precipitate anginal pain. Coronary artery spasm therefore *is said to be the mechanism for this pain. In support of this is the* observation that the pain is more likely to occur in cold environments, during emotional outbursts, after meals, and that it is relieved by vasodilators such as nitroglycerine and other nitrites. It may, however, be reproduced by a cellular metabolic disturbance independent of coronary spasm. Angina pectoris often antedates myocardial infarction. Certain physiologic observations, such as *the electrocardiographic disturbances* during the pain, are compatible with the idea of coronary artery spasm.

There is no known *pathologic* change characteristic of angina pectoris. Certain types of heart disease, however, are often associated with this syndrome.

The *clinical picture* typically consists of:

1. *Severe pain*, which has all of the characteristics of that described for coronary occlusion except (1) it does not last over fifteen minutes, (2) it is brought on by exercise, worry, or emotional disturbance such as fear, sorrow, etc., (3) it is relieved dramatically by nitroglycerine, and (4) it is relieved by rest.

2. There is usually no associated *circulatory collapse or shock*.

3. There is no *fever, leukocytosis* or increase in the *rate of the sedimentation* of erythrocytes.

4. There is *palpitation and dyspnea* during and for several minutes after the pain. Much of this is due to the fear of impending death. *Vertigo* and *faintness* are often associated with the pain, but *syncope* is rare.

5. The *electrocardiogram* shows depression of the *S-T* segments and lowering or inversion of the *T* waves during the attack. Characteristically *the electrocardiogram reverts to the previous pattern as soon as the pain* subsides. Since angina pectoris is not associated with infarction, there are no *QRS* changes of muscle death.

6. On *physical examination* few abnormalities are noted. During the episodes of pain, tachycardia, dyspnea, and elevation in blood pressure may be noted. Signs of previous cardiac disease are found in about 75 per cent of patients. A friction rub is never heard.

7. The *diagnosis* is made almost entirely by means of an adequate history. The *electrocardiographic* changes, when present, establish the diagnosis.

The *course* of angina pectoris is variable, depending on the nature of the *underlying cardiac state* and the adequacy with which the patient takes care of himself. The average *duration of life* after the onset of angina pectoris is about five to eight years. This is definitely longer in those patients who are properly managed and who abide by therapeutic instructions. The life expectancy, therefore, varies with different series of patients

less psychic and business strain, or environment preference The endocrine glands are known to influence arterial blood pressure, hypertension at the menopause is an example

The *pathologic picture* varies with the etiologic type of hypertension

amount of intercellular fibrous connective tissue and cells of chronic inflammation Although the left ventricle exhibits the greatest hypertrophy, the entire heart is usually involved This is especially true after left ventricular congestive heart failure has occurred and arteriolar disease becomes severe or there is a serious toxic state present, such as renal insufficiency

The arterioles and arteries reveal hypertrophy of the muscular coat. There m

this latter disease Since the changes are so diffuse, biopsy of the skeletal muscle is employed in diagnosis The process is patchy, however, and therefore se

If

wh

be present

TABLE 10 —ETIOLOGIC CLASSIFICATION OF DIASTOLIC HYPERTENSION

- I PRIMARY
  - A *Essential Hypertension*
    - 1 Benign
    - 2 Malignant
- II SECONDARY (may be clinically benign or malignant)
  - A *Renal*
    - 1 Hemorrhagic or glomerular nephritis
    - 2 Pyelonephritis
    - 3 Neoplasia
    - 4 Polycystic disease
    - 5 Nephroptosis
    - 6 Hydronephrosis
    - 7 Spinal bladder
    - 8 Aberrant renal artery
    - 9 Amyloidosis
    - 10 Infarction
    - 11 Trauma
    - 12 Congestive heart failure (?)
    - 13 Narrowing of lumen of renal artery
    - 14 Coarctation of aorta
  - B *Endocrine Dysfunction*
    - 1 Suprarenal
      - (a) Pheochromocytoma
      - (b) Adrenogenital syndrome
    - 2 Pituitary
      - (a) Basophilic syndrome
    - 3 Ovarian
      - (a) Menopausal
    - 4 Toxemia of pregnancy
  - C *Neuropsychologic*
    - 1 Psychoneurosis
      - (a) Anxiety neurosis
    - 2 Emotional disturbances
    - 3 Increased intracranial pressure
    - 4 Cerebral lesions
      - (a) Trauma
      - (b) Encephalitis
      - (c) Neoplasia
    - 5 Diencephalic syndrome (?)

In addition in renal hypertension there is an extra load on the heart damage to the myocardium by nephritis impaired renal function and associated toxic states. The student by glancing at the list of the various types of hypertension below can evaluate such combinations of injury from his knowledge of the disease and the resultant disturbed function.

The *etiologic factors* concerned in hypertension are unknown but the excellent work of Goldblatt Page Houssay and their associates should be studied by all students. Textbooks of physiology are also sources in this connection for discussions of the studies on hypertension and arterial blood pressure in general. These should be reviewed.

The arterial blood pressure is dependent upon

1 Cardiac output
2 Viscosity of the blood
3 Volume of the blood
4 Volume of the vascular bed
5 Peripheral resistance

The over-all pressure in a system of tubes containing flowing fluid obviously depends on the force with which it is being pumped and the rate at which it is being pumped. The more vigorous the pumping the higher the pressure and *vice versa*. Furthermore all other factors being equal the more viscous the flowing fluid the higher the pressure. It is understandable that if the volume of the vascular bed remains unchanged and the volume of blood placed in it is increased the pressure will rise. Blood vessels are elastic distensible tubes and since the physics of distensible tubes has received little or no attention by investigators the disturbances in hemodynamics in hypertension are not well understood. The resistance offered to the flow of fluid in tubes determines to a large extent the pressure within the tubes. Everyone has noted that opening the nozzle on a garden hose reduces the pressure of the water coming out. *In diastolic hypertension* the peripheral resistance at least is increased as evidenced by the arteriolar spasm and muscular hypertrophy in the arterioles.

In essential hypertension a diastolic hypertension all of the foregoing five factors are said to be within normal limits except the peripheral resistance which is increased. *The cause of the arteriolar spasm which* produces this is obscure.

The *incidence* and mortality of hypertension and hypertensive heart disease is high. There are over 100 000 deaths annually from hypertension in the United States where the incidence is highest among Negroes. Its occurrence throughout the world has not been properly studied. It occurs in both sexes with about equal frequency. Certain *hereditary or constitutional* factors seem to be important in influencing its incidence. High blood pressure tends to be more common in certain families especially people who are robust overweight and of a nervous emotional or excitable temperament. Climate may be ... to be less prevalent in tropical climates ... not be due to the climate itself *but to suc*... *tance*

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  - 6 [illegible]
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  - 9 [illegible]
  - 10 [illegible]
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The arterioles and arteries reveal hypertrophy of the muscular coat There may be intimal thickening and proliferation The walls of the arterioles are acutely inflamed with necrosis rupture and thrombosis in malignant essential hypertension The arteriolar pathology is diagnostic of this latter disease Since the changes are so diffuse, biopsy of the skeletal muscle is employed in diagnosis The process is patchy, however and therefore several sections of the muscle must be taken to detect the changes If there is true uremia associated with the hypertension the cardiac muscle will show a greater degree of degeneration Uremic pericarditis may also be present

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    - (b) Encephalitis
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  - 5 Diencephalic syndrome (?)

D *Toxic*
  1 Acute lead poisoning
  2 Systemic infections
  3 Toxemia of pregnancy

E *Miscellaneous*
  1 Visceral angiitides
    (a) Lupus erythematosus disseminatus
    (b) Periarteritis nodosa

The *clinical manifestations* of hypertensive heart disease are frequently drastically altered by the diseases associated with the hypertension other than the heart disease For example, the presence of renal insufficiency cerebral thrombosis or hemorrhage endocrine disturbances or gastrointestinal disturbances will modify the cardiac picture *Hypertension when fatal results* in death from

1 Heart failure
2 Renal failure
3 Cerebral thrombosis or hemorrhage

Not infrequently these exist simultaneously The cardiac manifestations themselves are essentially the same as those described for arteriosclerosis There may be disturbances in *cardiac function* due to

1 Congestive heart failure
2 *Angina pectoris*
3 Coronary occlusion
4 Cardiac irregularities and cardiac consciousness

The same discussions presented for the aging heart apply here In fact as in the aging heart the heart in hypertension is diseased because of

(a) Direct damage to the myocardium by toxins or disturbances in cellular metabolism
(b) Coronary insufficiency that is simultaneous angiospasm and impaired blood flow with increased cardiac work

It should also be remembered that many patients who have diastolic hypertension usually have arteriosclerosis and other manifestations of aging These patients describe a feeling of ill health with fatiguability weakness dyspnea on exertion cardiac pain of various sorts palpitation cardiac irregularities etc These vary in degree depending upon the duration and severity of the disease process Congestive heart failure angina pectoris or coronary thrombosis[1] are liable to occur These symptoms are the same as they are for the other etiologic types of similar heart disease Frequently renal disease with renal failure is present Then the symptoms and signs *of true uremia (drowsiness nausea vomiting* coma ammoniacal odor to the breath urea frost pericardial friction rub etc) modify the clinical cardiac picture

[1] Coronary artery occlusion resulting from an embolus in a coronary artery is extremely rare there being less than 50 such proved instances in the medical literature

Physical examination reveals the diastolic hypertension The ophthalmoscopic examination is an index of the extent of arteriolar disease and the severity of the hypertension and aids in differentiating benign from malignant hypertension The heart is usually large if the disease is well advanced Cardiac irregularities are observed premature contractions auricular fibrillation or flutter and paroxysmal tachycardia being noted most frequently Functional murmurs especially a blowing mitral murmur are usually found Aortic dilatation due to the hypertension produces aortic valvular insufficiency with a soft blowing diastolic aortic murmur All or some of the signs of left or left and right ventricular congestive heart failure may be encountered

The *blood pressure* varies a great deal normally to enable the circulation to meet the extreme variations in tissue demands for blood These variations may be even more ...
resting blood pressure
mm mercury systolic
it may be more prope
statistically for normal people However this may be equivalent to saying that arteriosclerosis is a *normal process* if found in a man over sixty years of age Such problems await solution In the young adult 135 and 90 may be the respective upper limits Still more important values which are little known are the levels reached during daily activities and experiences such as exercise emotional excitement eating disease and variations in environmental temperature and humidity Such stresses as these are probably responsible for temporary elevations in blood pressure which precipitate such syndromes as coronary occlusion cerebral hemorrhage and other vascular accidents It is because of these fluctuations that the pressure is maintained as near the resting levels as possible during treatment which provides rest and the avoidance of activities that elevate the arterial blood pressure

The *course* of hypertensive heart disease depends upon the type and severity of the hypertension the nature of the complicating states age of the patient ability and effort to follow therapeutic instructions meticulously etc Patients under good medical therapy live many years The physician must of course distinguish between the medical therapy he outlines for his patient and the actual regimen that results for this is important in prognosis Unless instructions are followed the cardiac complications congestive heart failure ...

grave and
se of death

course
practically all patients
established

D *Toxic*

1 Acute lead poisoning
2 Systemic infections
3 Toxemia of pregnancy

E *Miscellaneous*

1 Visceral angiitides
(*a*) Lupus erythematosus disseminatus
(*b*) Periarteritis nodosa

The *clinical manifestations* of hypertensive heart disease are frequently drastically altered by the diseases associated with the hypertension other than the heart disease. For example, the presence of renal insufficiency, cerebral thrombosis or hemorrhage, endocrine disturbances, or gastrointestinal disturbances will modify the cardiac picture. *Hypertension when fatal results* in death from

1 Heart failure
2 Renal failure
3 Cerebral thrombosis or hemorrhage

Not infrequently these exist simultaneously. The cardiac manifestations *themselves are essentially the same as those described for arteriosclerosis.* There may be disturbances in *cardiac function* due to

1 Congestive heart failure
2 Angina pectoris
3 Coronary occlusion
4 Cardiac irregularities and cardiac consciousness

The same discussions presented for the aging heart apply here. In fact as in the aging heart, the heart in hypertension is diseased because of

(*a*) Direct damage to the myocardium by toxins or disturbances in cellular metabolism
(*b*) Coronary insufficiency, that is, simultaneous angiospasm and impaired blood flow with increased cardiac work

It should also be remembered that many patients who have diastolic hypertension usually *have arteriosclerosis and other manifestations of aging.* These patients describe a feeling of ill health with fatiguability, weakness, dyspnea on exertion, cardiac pain of various sorts, palpitation, cardiac irregularities, etc. These vary in degree depending upon the duration and severity of the disease process. Congestive heart failure, angina pectoris or coronary thrombosis[1] are liable to occur. These symptoms are the same as they are for the other etiologic types of similar heart disease.

Frequently renal disease with renal failure is present. *Then the symptoms and signs of true uremia* (drowsiness, nausea, vomiting, coma, ammoniacal odor to the breath, urea frost, pericardial friction rub, etc.) modify the clinical cardiac picture.

[1] Coronary artery occlusion resulting from an embolus in a coronary artery is extremely rare, there being less than 50 such proved instances in the medical literature.

nervous system must be made in the early stages of the disease before the pathologic processes have become irreversible if satisfactory results are to be obtained. Its value has yet to be established. The newer vasodilator drugs seem to have eliminated sympathetic surgical procedures for hypertension.

The treatment of the *cardiac complications*, congestive heart failure, *angina pectoris*, *coronary* occlusion, cardiac irregularities, etc., have been described elsewhere. The management of these should supplement the ... *Prevention* of cardiac damage is attained ... ons ... rist

It is beyond the intention of these ... e of the management of the many noncardiac complications of hypertension. The student should obtain this from other more detailed sources in the medical literature.

SYPHILITIC HEART DISEASE

Syphilis produ... of the coronary ... aortic aneurysm, and (4) rarely, infection of the cardiac musc... per se ...

syphilis is becoming rare and ... *Treponema pallidum* is the et... heart disease. The majority ... and fifty-five years, but the age of patients with this disease ... from less than ten to more than seventy years. *Men* are more often affected *than women*, the ratio being about 3 to 1. The *economic and social status* is important, since people of the low income and low social levels are more likely to contract and neglect syphilitic infections. This is true throughout the world.

In brief, the *pathologic* changes occur because:

1. *Involvement of the coronary vessels* occurs with narrowing of their lumina and resultant impairment of blood supply to the myocardium. This results from direct involvement of the anterior and posterior coronary arteries near their origins. The coronary ostia may become narrowed or even occluded by local aortitis at the root of the aorta.
2. *Aortitis* near the sinuses of Valsalva results in dilatation of the aortic ring and fibrosis and distortion of the aortic valve. These anatomic changes are followed by *aortic* regurgitation, which in turn produces ... work for the left ventricle and decreases ...ing diastolic blood pressure.

TABLE 11 —THE CAUSE OF DEATH IN ESSENTIAL HYPERTENSION

	Per cent
Congestive heart failure	45
Cerebral artery hemorrhage or thrombosis	19
Coronary failure	16
Renal failure	9
Miscellaneous	11

The *treatment* of hypertensive heart disease may be divided into

1 Treatment of the hypertension
2 Prevention or delay of cardiac damage
3 Management of the cardiac disease and its complications
4 Management of the various noncardiac complications due to the hypertension

Hypertension cannot be cured today nor is there any specific therapy for essential hypertension The sooner the doctor and the patient realize this the more effective therapy will become For therapy to be effective a *complete inventory of the patient's health* must be obtained in order to deter

cause reversible

Therapy should

and rigorously

It should include

1 *Mental and physical rest* which are extremely important All anxiety states should be vigorously treated to effect good results
2 The *diet* should be well balanced in every respect but should not include an excessive amount of proteins preferably not more than 1 gram per kilogram of body weight Water should be permitted freely but *sodium* in all its forms should be restricted
3 *Sedatives* phenobarbital (gr 1 or 60 mg) should be given as often as is necessary to obtain the desired results Remember that the response of patients to drugs varies widely
4 *Vasodilators* Most of them are too toxic to be of continuous service in a disease as chronic as hypertension Anyone who offers a cure for hypertension should be approached with great caution and skepticism The new vasodilators such as hexamethonium and hydrazinophthalazine offer considerable promise but have yet to be evaluated thoroughly More effective drugs of this nature are being developed rapidly
5 *General hygienic measures* such as laxatives vacations rest in bed after meals plenty of sleep and prompt care of infections are to be urged
6 *Frequent visits to the physician* are necessary for follow up and most important to keep the patient on his medical regimen and reminded that his illness requires continual treatment
7 *Surgical therapy* for essential hypertension has a limited role at present It should be employed early and only after *adequate medical therapy* has failed A surgical approach to the sympathetic

elevation of the systolic and severe depression of the diastolic blood pressure (Fig 144) This high pulse pressure is associated with the following signs

a Corrigan or water hammer pulse is a pulse which is full and bounding It is best elicited by raising the patient's arm vertically while palpating the radial artery with the palmar aspects of the four fingers

b Traube's pistol-shot sound is a loud sound heard with each systolic ejection over the femoral artery or any large artery which is partially compressed by the bell of the stethoscope

c Capillary pulsations are elicited in several ways

(1) A *finger nail*
area is produc
area to reduc
diastole

(2) The lighted end of a small *pocket flashlight* is pressed against the finger pad revealing a red blush which pulsates in intensity with each cardiac systole

(3) A clean glass *microscope slide* is pressed against the skin over the forehead until a small white blanched area is produced The blanched area decreases in size with cardiac systole and increases with diastole

(4) The forehead is *rubbed vigorously* until an area of erythema is produced This flushed area will pulsate in intensity with each cardiac cycle

d D[illegible]

compressed area of a large artery due it is contended to the reversal of blood flow during diastole

[illegible] produced by the Treponema results in a clinical syndrome which is not easy to identify The important diagnostic characteristics are

a Positive evidence of [illegible] [illegible]
about 100 per ce[illegible]

b Dyspnea [illegible]

c Substernal pain ranging from a vague type to the true pain of angina pectoris

d [illegible] This is heard best

e

4 [illegible] aneurysms will produce rather characteristic manifestations The aneurysms which localize near the root or ascending aorta are more apt to produce *signs* whereas those of the arch produce early symptoms Aneurysms produce clinical disturbances by

a Pressure phenomena due to their mass and position
b Disturbances in hemodynamics
c Hemorrhage

3 *Aortitis* may also result in weakening of the wall with formation of aneurysm An aneurysm of the root of the aorta usually interferes with coronary circulation and may interfere mechanically with cardiac function

4 There may be *diffuse inflammation of the myocardium* produced by direct spirochetal infection This is rare and has been doubted by some observers The clinical picture is essentially the same as that previously described for myocarditis

5 *Spirochetal infection* of the cardiac muscle may be localized with *gumma* formation The clinical picture is determined by the location of the gumma for example a gumma involving the H node will result in complete heart block and the like

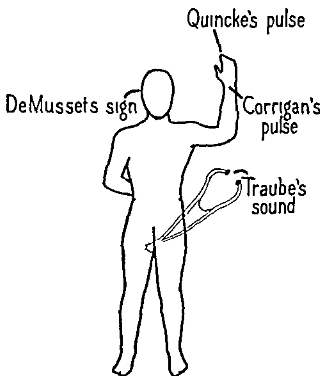


FIG 144 —Illustration summarizing the important peripheral vascular signs of aortic valvular insufficiency

The student is urged to consult sources describing in detail the pathologic changes produced in the heart and aorta by the *Treponema pallidum*

The *clinical picture* depends upon the pathologic changes which produce the cardiac disease (1) *Involvement of the coronary vessels and their ostia* with narrowing of their lumina may result in angina pectoris or even on rare occasions coronary occlusion (2) *Aortic valvular insufficiency* often leads to congestive heart failure The associated murmurs and thrills have already been described Some of the largest hearts encountered clinically occur as a result of this type of involvement Aortic insufficiency produces

elevation of the systolic and severe depression of the diastolic blood pressure (Fig 144) This high pulse pressure is associated with the following signs

a Corrigan or *water hammer pulse* is a pulse which is full and bounding [illegible] while palpating the

[illegible] with each systolic ejection over the femoral artery or any large artery which is partially compressed by the bell of the stethoscope

c *Capillary pulsations* are elicited in several ways

(1) A *finger nail* is pressed down at its edge until a small blanched area is produced under it *Capillary* pulsations cause this blanched area to reduce in size with cardiac systole and to increase with diastole

(2) The lighted end of a small *pocket flashlight* is pressed against the finger pad revealing a red blush which pulsates in intensity with each cardiac systole

(3) A clean glass *microscope slide* is pressed against the skin over the forehead until a small white blanched area is produced The blanched area decreases in size with cardiac systole and increases with diastole

(4) The forehead is *rubbed vigorously* until an area of erythema is produced This flushed area will pulsate in intensity with each cardiac cycle

d [illegible]

[illegible] of a large artery due it is contended to the reversal of blood flow during diastole

3 *Acute aortitis* produced by the *Treponema* results in a clinical syndrome which is not easy to identify The important diagnostic characteristics are

a *Positive evidence of syphilis* The serologic reactions are positive in about 100 per cent of patients with acute aortitis

b *Dyspnea on exertion* much like that previously described

c *Substernal pain* ranging from a vague type to the true pain of angina pectoris

d *A tambour quality* and accentuated aortic second sound This is heard best when the patient stops his breathing in deep expiration

e *Roentgenographic* evidence of dilatation of the aorta

4 *Syphilitic aneurysms* will produce rather characteristic manifestations The aneurysms which localize near the root or ascending aorta are more apt to produce *signs* whereas those of the arch produce early symptoms Aneurysms produce clinical disturbances by

a Pressure phenomena due to their mass and position

b Disturbances in hemodynamics

c Hemorrhage

The nature of the pressure phenomena is determined by the *size, location and rapidity of development* of the aneurysm. An aneurysmal mass which does not disturb other structures by pressure will produce little trouble unless it ruptures. It is impossible in any single monograph to describe all of the possible symptoms and signs due to an aneurysm. The disturbances are logical if the student correlates anatomic relationships and attempts to predict the results that a growing pulsating mass of varying size could produce if it developed from various portions of the aorta. For example, an aneurysm located in the ascending portion of the aorta might result in pressure on the sternum. Pulsating pressure on the sternum causes pain and later erosion of the sternum with a protruding tender pulsating mass.

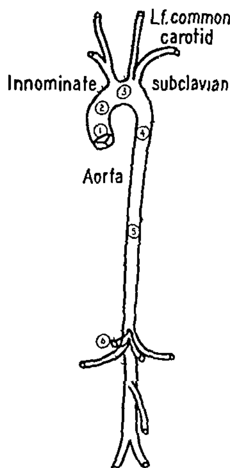


Fig. 145.—Diagram of the common sites of an aortic aneurysm. When located at points 1 and 2 signs of an aneurysm are easily discernible since it is closer to the thoracic [illegible] regions 3 and 4 are more prone to produce symptoms by pressure effects.

under an inflamed area of skin This mass is prone to rupture An aneurysm of the arch of the aorta may press on the left recurrent laryngeal nerve with resulting hoarseness (Fig 145) If this aneurysm grows, it will obviously produce pressure on the trachea or left bronchus The constant ... cartilaginous tubes is soon followed by inflam-

[illegible]

to chest ... for pneumonia to develop in the pulmonary area supplied ... There may be a sudden and extensive rupture into the *bronchus or trachea* with ensuing death The aneurysm may compress the *esophagus* and lead to impairment of swallowing A fatal hemorrhage into the esophagus may occur An aortic aneurysm may rupture into the pulmonary artery,

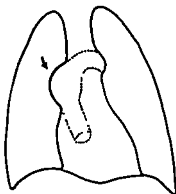


Fig 146 The solid line outlines the cardiac silhouette in the anteroposterior view of a patient with an aneurysm at the root of the aorta

producing acute anastomosis between these two vessels and resulting in a ... atelectasis pneumonia ...

[illegible]

aneurysms

An aneurysm will disturb the flow and pressure of blood in distal arteries therefore a properly placed aneurysm may reduce the pressure in the left brachial artery without disturbing that in the right thereby producing a wide difference in the arterial pressures in the two arms An aneurysm of the root of the aorta may sometimes impair the coronary circulation resulting in the coronary pain

The nature of the pressure phenomena is determined by the *size, location and rapidity of development* of the aneurysm. An aneurysmal mass which does not disturb other structures by pressure will produce little trouble unless it ruptures. It is impossible in any single monograph to describe all of the possible symptoms and signs due to an aneurysm. The disturbances are logical if the student correlates anatomic relationships and attempts to predict the results that a growing pulsating mass of varying size could produce if it developed from various portions of the aorta. For example an aneurysm located in the ascending portion of the aorta might result in pressure on the sternum. Pulsating pressure on the sternum causes pain and later erosion of the sternum with a protruding tender pulsating mass.

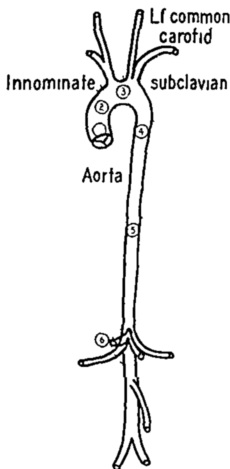


FIG. 1 —D[illegible] of the [illegible] sites of an a[illegible] aneurysm. When located at point [illegible]

under an inflamed area of skin. This mass is prone to rupture. An aneurysm of the arch of the aorta may press on the left recurrent laryngeal nerve with resulting hoarseness (Fig. 143). If this aneurysm grows it will obviously produce pressure on the trachea or left bronchus. The constant pulsation against these cartilaginous tubes is soon followed by inflammation, erosion and bleeding from the mucosa with mild or severe *hemoptysis*. If there is enough compression and edema of the bronchial mucosa to close the lumen, *atelectasis* will develop. There is a strong tendency for pneumonia to develop in the pulmonary area supplied by the bronchus. There may be a sudden and extensive rupture into the *bronchus or trachea* with ensuing death. The aneurysm may compress the *esophagus* and lead to impairment of swallowing. A fatal hemorrhage into the esophagus may occur. An aortic aneurysm may rupture into the pulmonary artery,

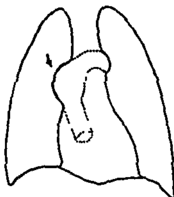


Fig. 143.—The solid line outlines the cardiac silhouette in the anteroposterior view of a patient with an aneurysm at the root of the aorta.

producing acute anastomosis between these two vessels and resulting in a syndrome typical of *patent ductus arteriosus*. When atelectasis, pneumonia or recurrent laryngeal paralysis occurs, the physical signs characteristic of these pathologic states are evident on exam ...

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Treatment.—The *treatment* of thoracic aneurysms is still unsatisfactory and mainly supportive. *Rest, sedatives, morphine* and *penicillin* therapy are essentially all that one can offer. *Proper hygienic and psychic measures* are indicated, as they are in all problems of therapy.

The *diagnosis* of syphilitic heart disease is usually not difficult. Clinical evidence of heart disease that is usually syphilitic in origin, such as aortic insufficiency, aortitis or aneurysm of the aorta in a patient who is known to have syphilis, may be confirmed by a history of a primary lesion, adequate previous diagnosis in a patient inadequately treated, or conclusive serologic evidence for a number of years establishes the diagnosis. The infection must be present about ten or more years before aortic insufficiency or aortic aneurysm develops, whereas aortitis appears clinically after only several weeks of infection.

The *course* of cardiovascular syphilis varies with the extent and duration of the disease. *Syphilitic aortitis* will respond to therapy with complete cure if early and adequate antisyphilitic therapy is administered. The course of *aortic insufficiency and aneurysm* is determined not by the syphilis but by the heart disease. Both of the latter are extremely grave. Once *congestive heart failure* has occurred in aortic insufficiency, the majority of patients will be dead within two years. Cardiovascular syphilis carries a grave prognosis. *Angina pectoris, coronary occlusion* and *congestive heart failure, ruptured aneurysm* and *pneumonia* are the most common complications of cardiovascular syphilis.

The *treatment* of syphilitic heart disease resolves itself into two categories: (1) *treatment of heart disease* and (2) *treatment of the syphilis*.

The *treatment of the heart disease always takes precedence* over the syphilitic infection. The heart disease should be managed like any similar type of heart disease and as though the patient had no syphilitic infection. This entails proper care of congestive heart failure, angina pectoris or irregularities if present, as outlined elsewhere in the compendium.

The *treatment of the syphilis* consists in the use of penicillin which is administered while the patient is being treated for his heart disease. Although penicillin is usually recommended in doses of 600,000 units daily for 10 days, it is preferable to employ 1,000,000 units daily for 10 days. This should be repeated if the titer of the quantitative Kahn or Kolmer does not decline. In fact, the course of penicillin could be repeated in a couple of weeks anyway. There is much discussion about the dangers due to Herxheimer reactions, especially during the acute cardiovascular episodes. Some physicians, on the other hand, have used penicillin prophylactically in all of their patients with congestive failure and have learned later that the patient has syphilis but have observed no untoward reactions. This has happened often enough to indicate absence of danger from reactions other than febrile to penicillin therapy. *Paradoxical* therapeutic reactions have not been shown to occur with the use of penicillin nor has any cardiac state been materially, if at all, aggravated by penicillin. Do not hesitate to use penicillin and do not use it with economy. *The heavy metals no longer have any place in antisyphilitic therapy.*

RHEUMATIC HEART DISEASE

Rheumatic fever is characteristically associated with involvement of the heart. The endocardium, myocardium and pericardium may be involved individually or all three simultaneously. The cardiac disease varies considerably in degree and frequency, being more prevalent and severe in the cold regions of the earth than in the warm ones. The fulminating types of acute rheumatic heart disease seen in the New England States are relatively rare in the Gulf Coast States. Rheumatic fever of a subtle sort is *extremely common in warm climates*.

The *etiology* of rheumatic fever *has not been established*, nor has the pathogenetic relationship of rheumatic fever to the cardiac state. It is known that streptococcic infections, particularly of the respiratory tract, are related to acute rheumatic fever. The epidemiology, mode of entry, precipitating causes, and influence of heredity are not well understood. There is no significant difference in *incidence by sex*. Acute rheumatic fever is much more frequent in the young *age* group (Table 12), particularly in children from four to fifteen years of age. It is common in the young adult and rare in the old patient. Rheumatic heart disease begins during illness from acute rheumatic fever, although it may not be noted until many years later. There is no significant *racial* difference in incidence. Rheumatic fever seems to have a *familial tendency* which is probably more environmental than genetic. Poor housing, nutrition, and medical care and crowded surroundings appear to be mainly concerned with the familial incidence. Economically and socially poor families tend to remain so for generations.

TABLE 12 — THE INCIDENCE OF RHEUMATIC HEART DISEASE BY AGE

Years	Per cent
0–10	12
10–20	31
20–30	14
30–40	17
40–50	13
50–60	8
60–70	4
Over 70	1

The path[illegible]
func[illegible]
lesi[illegible] process manifested by an accumulation around small vessels of small round mononuclear cells, a few giant cells and several rather large polymorphonuclear cells. There may be many such coalescing nodules or isolated solitary *ones*. These nodules heal with *scar or fibrous tissue formation*. The Aschoff body is apparently preceded by a small area of *necrosis* with a *hemorrhagic tendency*. These pathologic processes develop in the myocardium whenever the heart is involved. There may be associated endocardial or pericardial damage as well. When all three structures are involved, pancarditis exists.

Treatment—The *treatment* of thoracic aneurysms is still unsatisfactory and mainly supportive *Rest sedatives morphine* and *penicillin* therapy are essentially all that one can offer *Proper hygienic and psychic measures* are indicated as they are in all problems of therapy

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RHEUMATIC HEART DISEASE

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The *endocardium* may be involved in any area. The valvular leaflet most often shows a *verrucous* inflammatory reaction. The verrucal lesions or vegetations consist essentially of thrombi, that is, fibrin, erythrocyte and leukocytes. These heal with fibrous tissue formation, thickening and distortion of the valves, and consequent *impaired valvular function*. Valvular function is even more disturbed when the *chordæ tendineæ* are involved. *The descending order of frequency of valvular involvement is* essentially as follows

1 Mitral
2 Mitral and aortic
3 Aortic
4 Mitral, aortic and tricuspid
5 Tricuspid
6 Pulmonic

The *myocardial inflammation* varies in degree. When it is severe and diffuse, acute myocarditis results, and the clinical and pathologic picture as described previously for myocarditis in general is present. The Aschoff bodies may be widely distributed and isolated. Their localization in the AV node results in heart block, and their localization in one or the other bundle branches produces bundle branch block.

The *pericardium* is involved in the process relatively often. This is manifested by acute fibrinous pericarditis, then pericarditis with effusion, and finally chronic adhesive pericarditis. These processes have been discussed previously.

The *clinical manifestations* of rheumatic heart disease include

1 Those associated with the episodes of acute rheumatic fever
2 Those resulting from the scars and fibrosis of healed lesions

Acute rheumatic heart disease, in turn, consists in essentially two groups of syndromes

1 The *general or systemic manifestations* of acute rheumatic fever
2 The *cardiac manifestations themselves*

The student should acquaint himself with the clinical picture of acute rheumatic fever in all its manifestations. Acute rheumatic fever is characterized essentially by (1) *acute migratory polyarthritis (swollen, inflamed,* tender and painful joints which return to normal rapidly, with previously normal joints becoming involved; this continues for days or weeks), (2) fever (100° to 105° F or more) which may be remittent or intermittent, (3) chills or chilly sensations, (4) headache, (5) malaise, weakness and prostration, (6) chorea, (7) sweating, (8) dyspepsia (anorexia, nausea, vomiting), (9) epistaxis, (10) loss of weight, (11) erythema multiforme or purpuric rash, and (12) subcutaneous nodules in the fascial planes and tendons. The clinical picture may be severe or so mild as to manifest itself merely by fatigue and low grade fever. Antecedent upper respiratory tract infections and acute or chronic tonsillitis or paranasal sinusitis usually exists. Leukocytosis (10 000 to 20 000), increase in the rate of sedimentation of the erythrocytes, increase in the antistreptolysin titer, etc., will be noted on laboratory examination.

The *cardiac involvement* is manifested in the *acute disease* by

1 *Tachycardia* out of proportion to the fever
2 *Palpitation*
3 *Precordial aching* soreness tenderness or heart consciousness
4 *Dyspnea* on exertion
5 Symptoms and signs of *severe congestive heart failure* in advanced forms of myocarditis
6 Murmurs of varying severity, particularly the *murmurs of mitral* or aortic insufficiency There may be a rumbling diastolic mitral *murmur of relative mitral stenosis* if the left ventricle is greatly dilated An aortic systolic murmur is heard if there are verrucae on the aortic cusps A pericardial friction rub may be heard
7 *Electrocardiographic abnormalities* in the *QRS complexes* and *T* waves and prolongation or variations in duration on serial tracings of the *P-R* interval The changes of pericarditis may be noted
8 *Roentgenologic* changes in cardiac configuration or size in the mild episodes and diffuse enlargement in the severe ones Pericardial effusion may be noted roentgenologically

The *cardiac manifestations of chronic rheumatic heart disease* are of three types

1 Symptoms and signs of *valvular heart disease* (discussed previously) of which the following are the most common in descending order of frequency

(*a*) Mitral insufficiency
(*b*) Mitral stenosis
(*c*) Mitral insufficiency and aortic stenosis
(*d*) Mitral stenosis and aortic insufficiency
(*e*) Tricuspid insufficiency or stenosis and any combination of, or all of, the foregoing valvular lesions

2 Chronic *mediastinopericardial* adhesions or constrictive *pericarditis* (concretio cordis)

3 Chronic *myocardial damage* or scarring This is characterized by the clinical manifestations described previously for myocarditis and *includes* particularly disturbances in impulse conduction Bundle branch *block* AV block and *T* wave changes are among the most common manifestations of old areas of fibrosis

Congestive heart failure is especially likely to develop as a result of any of these lesions Angina pectoris and auricular flutter and fibrillation are frequently present with mitral *aortic stenosis*

The course of *acute rheumatic heart disease* varies considerably Most of the patients survive the *illness* Those who have repeated episodes of rheumatic fever and especially those who *are improperly* managed are prone to develop chronic rheumatic heart disease valvular heart disease in particular Most *patients* who have had mild or even severe *rheumatic* fever and cardiac involvement make a complete clinical recovery Those who sustain large areas of fibrosis or valvular sclerosis retain these lesions throughout the remainder of *their lives* Infections chronic illness cardiac

damage from other causes (poor nutrition, anemia, hypertension, etc.), physical overexertion and severe psychic disturbances are prone to precipitate heart failure or severe cardiac irregularities such as auricular fibrillation. Congestive heart failure and subacute bacterial endocarditis are severe complications which often result in death. Severe valvular disease and chronic congestive heart failure are causes for invalidism. Pneumonia or severe systemic infections are complications which often result in death. Under proper care, however, some patients live practically normal lives until the seventh or eighth decade of life with severe valvular disease or pericardial adhesions.

Treatment.—The treatment of rheumatic heart disease varies with the severity and stage of the disease. The main objective is to *prevent rheumatic fever*. (Consult textbooks of medicine and monographs for details.) Prolonged daily administration of sulfadiazine and the use of penicillin during periods of respiratory infections for ten- to fifteen-day intervals have been recommended and may be tried. Warm climates may also assist, but all of these procedures do not necessarily offer much general promise nor can one know which patients are particularly likely to be benefited. If acute rheumatic fever has developed, the aim becomes that of preventing or reducing cardiac damage. During this time *bed rest, good food*, plenty of *liquids* and *good nursing* attention are of paramount importance. *Salicylates* in full doses must be administered (1 grain or 60 mg. per pound of body weight, or to tolerance). The value of ACTH and cortisone remains to be fully established. If salicylates fail to function adequately, these newer *drugs may be tried in the severe acute phases of the disease*. They have not been shown to prevent scar formation.

The patient must remain in *bed* until all systemic signs of activity of the acute rheumatic fever have subsided, as evidenced by:

1. Normal body temperature
2. Normal resting cardiac rate
3. Normal sedimentation rate
4. Normal blood picture
5. *No symptoms*

Even when these criteria have been fulfilled, bed rest should not be suddenly terminated. The patient should return to normal activity slowly. Should there be any exacerbation of the rheumatic fever, he should return *to bed promptly*. *Measures to prevent a recurrence of rheumatic fever* are to be continued throughout the patient's life. *Frequent medical examinations* with cardiac surveys are indicated. *Tonsillectomy* is indicated *only* if the tonsils are diseased.

Patients with *chronic valvular disease* or *chronic pericardial disease* should:

1. *Rest* (physically and mentally)
2. *Work indoors* preferably
3. Receive prompt *treatment for all infections*
4. *Avoid competitive sports* or strenuous exercise, the degree of exercise permitted being determined by the clinical picture

5 Follow *good hygienic principles* of diet sleep etc
6 Receive adequate treatment for *complications* as they arise
7 Receive *surgical* attention for chronic pericardial adhesions when necessary

Surgical relief of the obstruction due to mitral stenosis is of considerable therapeutic value Properly trained surgical teams can perform commissurotomies with relatively little operative risk to the patient and with considerable relief from his cardiac difficulties The criteria for selection of patients for surgery will not be discussed here a competent cardiologist is aware of these Only rarely if *ever* should he have to resort to cardiac catheterization and then only if congenital lesions of the heart are suspected as complicating defects

[illegible] attacks of acute rheumatic fever may be [illegible]

orally 250,000 units twice daily [illegible] stead of the sulfadiazine The relative merits of the [illegible] two drugs or the full value or either remain to be established

THYROID HEART DISEASE

[illegible] function may result in heart disease [illegible] thyroid heart disease and (2) hypo- [illegible] neglected but if treated early and properly they are completely reversible For this reason it is essential that thyroid disease be recognized early before irreversible cardiac damage occurs

Hyperthyroid Heart Disease

[illegible]

adult *age* group since hyperthyroidism is more frequent at this age This is particularly true of Graves disease Toxic adenoma is more likely to produce hyperthyroid heart disease in the later (forty to fifty years) age group It is well to remember however that the disease may occur at any age

The *sex* difference in incidence is considerable The *female* sex is involved five times as often as the male There are *no racial* differences

The *etiologic* mechanism for thyrotoxic heart disease remains mainly conjectural There are certain factors however which must contribute to some extent to the development of the cardiac damage

damage from other causes (poor nutrition, anemia, hypertension, etc.), physical overexertion, and severe psychic disturbances are prone to precipitate heart failure or severe cardiac irregularities such as auricular fibrillation. Congestive heart failure and subacute bacterial endocarditis are severe complications which often result in death. Severe valvular disease and chronic congestive heart failure are causes for invalidism. Pneumonia or severe systemic infections are complications which often result in death. Under proper care, however, some patients live practically normal lives until the seventh or eighth decade of life with severe valvular disease or pericardial adhesions.

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The patient must remain in *bed* until all systemic signs of activity of the acute rheumatic fever have subsided, as evidenced by:

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Even when these criteria have been fulfilled, bed rest should not be suddenly terminated. The patient should return to normal activity slowly. Should there be any exacerbation of the rheumatic fever, he should return to bed promptly. Measures to prevent a recurrence of rheumatic fever are [illegible] s life. *Frequent medical examina-* [illegible] *Tonsillectomy* is indicated *only* if [illegible]

Patients with *chronic valvular disease* or *chronic pericardial disease* should:

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2. *Work indoors* preferably.
3. Receive prompt *treatment for all infections*.
4. *Avoid competitive sports* or strenuous exercise, the degree of exercise permitted being determined by the clinical picture.

The *electrocardiogram* is of considerable value in detection of myocardial damage There are no characteristic electrocardiographic changes in hyperthyroidism although the tachycardia and P and T waves of high ... e study is important for detec-

propylthiouracil, or iodine is of great diagnostic importance, iodine alone being employed infrequently today

Rheumatic fever essential hypertension neurocirculatory asthenia and thyroid crisis are among the clinical states which are concerned in the

by the duration
Early adequate
there are compli-
cating cardiac disturbances such as arteriosclerosis of the coronaries the prognosis is worse All patients should be cured Only those who fail to see a physician early enough or who have underlying associated cardiac disease should have an unfavorable prognosis

Treatment —The treatment of hyperthyroid heart disease consists in management of

1 The *hyperthyroid disease*
2 The *heart disease per se*

sedatives
propylthiouracil and other measures employed for the hyperthyroidism Quinidine and or digitalis is *indicated* for the paroxysms of flutter fibrillation and tachycardia if they become annoying *Digitalis* and other measures already out... e to be used when failure develops T... ures much more digitalis than the us... that the patient be classified in Functional Class I or at least in Class II before thyroidectomy is undertaken Prolonged use of propylthiouracil may be necessary to effect the functional objective

Hypothyroid Heart Disease

The hypo

1. The *increased metabolism* throughout the body increases the demand on the heart and circulation. This in turn increases cardiac work.
2. The *increased cellular metabolism* in the cardiac muscle probably also suffers from the toxic state influencing the entire body.
3.

output and work.

The *pathologic* pattern of the cardiac lesion is not characteristic. It consists essentially of hypertrophy and dilatation with degenerative change which vary from the usual mild forms to necrosis of the myocardium in some instances.

The *clinical manifestations* include those manifestations due to the

1. Hyperthyroidism
2. Heart disease itself

The *cardiac manifestations* consist of palpitation, dyspnea on exertion and precordial discomfort. Paroxysms of auricular flutter, auricular fibrillation and tachycardia are frequent. These forms of tachycardia plus the concomitant sinus tachycardia are responsible for the palpitation. In addition, *the forceful, vigorous "pounding" or beating of the heart produces* palpitation. Angina pectoris is not uncommon.

Examination of the cardiovascular system will reveal sinus tachycardia, cardiac enlargement, functional mitral systolic murmur and vigorous pulsations of the visible arteries to be particularly prominent. The cardiac arrhythmias may be found. The *blood pressure* is characterized by a high *systolic pressure* due to the vigorous and rapid ejection of blood from the ventricles, and a normal or low *diastolic blood* pressure due to reduced peripheral resistance from peripheral vascular dilatation. The *pulse pressure* is therefore high. The high pulse pressure may result in capillary pulsations, water hammer or Corrigan pulse and the other vascular signs encountered in aortic insufficiency. The symptoms and signs of *left and right congestive heart failure* develop when the cardiac reserve diminishes.

The *diagnosis* of hyperthyroid heart disease is not difficult. The presence of definite evidence of heart disease in a patient with hyperthyroidism with or without any other cause for heart disease establishes the diagnosis of hyperthyroid heart disease. The other etiologic agents for heart disease, if present, contribute in various ways to the cardiac status.

The *diagnosis of hyperthyroidism* is established by the presence of weight loss, definite increase in appetite, weakness, the cardiovascular manifestations mentioned previously, restlessness and excitability, fine tremor, sweating, warm, flushed and fine textured skin, the ophthalmic signs of Graves' disease, goiter, systolic bruit over the thyroid, sensitiveness to heat, increase in the BMR, decrease in blood cholesterol concentration and an increased blood serum iodine (over 8 gamma per 100 cc). The rate of excretion in the urine and quantitative deposition in the thyroid gland of a tracer quantity test dose of I^{131} can be of considerable value in the diagnosis of hyper- and hypothyroidism. This must be done *accurately* to be reliable and useful.

The *electrocardiogram* is of considerable value in detection of myocardial damage There are no characteristic electrocardiographic changes in ... and *P* and *T* waves of high ... study is important for detec-

propylthiouracil or *iodine* is of great diagnostic importance iodine alone being employed infrequently today

Rheumatic fever essential hypertension neurocirculatory asthenia and thyroid crisis are among the clinical states which are concerned in the

... the duration ... early adequate ... If there are compli- ... the coronaries the ... Only those who fail to see a physician early enough or who have underlying associated cardiac disease should have an unfavorable prognosis

Treatment The treatment of hyperthyroid heart disease consists in management of

1 The hyperth...

... ment at subsequent surgery is sufficient to produce permanent cure ...

Many ...

... sedatives ... other measures employed for the hyperthyroidism Quinidine and or digitalis is indicated for the paroxysms of flutter fibrillation and tachycardia if they become annoying *Digitalis* and other measures already outlined for congestive heart failure are to be used when failure develops The hyperthyroid patient often requires much more digitalis than the usual cardiac patient It is advisable that the patient be classified in Functional Class I or at least in Class II before thyroidectomy is undertaken Prolonged use of propylthiouracil may be necessary to effect the functional objective

Hypothyroid Heart Disease

... largement and dilatation The hypothyroid state often follows thyroidectomy for thyroid disease

Hypothyroid heart disease occurs at any *age*, being most frequent in the young and middle-aged adult. There is no relationship to *sex* except that more instances of postoperative hypothyroid heart disease occur in women since more women undergo thyroidectomy.

The *pathologic* picture is not characteristic. There is cardiac hypertrophy and dilatation with various degrees of muscular degeneration. The interstitial fluid is increased. There is also a great tendency for accumulation of pericardial fluid.

The *clinical manifestations* entail those of

1. *Hypothyroidism*
2. *Heart disease*

The *heart disease* is manifested by dyspnea, relatively slow pulse, functional systolic mitral murmur and cardiac enlargement. There may be manifestations of congestive heart failure if the disease has existed for a long time and the hypothyroidism is relatively pronounced. The *electrocardiogram* is fairly characteristic in that all complexes are of low amplitude, particularly the *T* waves. The roentgenographic study shows cardiac enlargement.

The *hypothyroidism* is manifested by impairment of appetite, gain in weight, mental and physical sluggishness, dry, thick and coarse skin and hair, yellowish pasty color to the skin, impairment of growth and thick tongue in the cretin, sensitiveness to cold, low BMR, high serum cholesterol, low bound blood iodine (less than 3 gamma per 100 cc). The I^{131} uptake by the thyroid gland and urinary excretion can be diagnostically useful if done well.

The *course* of the heart disease depends upon the severity and duration of the hypothyroidism. If the patient is treated early and adequately, the cardiac state is *reversible*. *Sudden anginal failure* with death may occur in a patient who is treated too rapidly with thyroid extract.

Treatment. The treatment of hypothyroid heart disease consists entirely in treatment of the hypothyroidism by thyroid extract. This is accomplished by the oral administration of $\frac{1}{4}$ to $\frac{1}{2}$ grain (15 to 30 mg.) of thyroid extract daily to the patient initially, then increasing the dose over a period of four to six weeks. These patients should be watched carefully for, and warned of, anginal pain from too rapid thyroid extract therapy. If congestive heart failure exists, it should be treated as previously outlined, although digitalis is of little benefit in most instances.

CONGENITAL CARDIAC ANOMALIES

The congenital anomalies of the heart are relatively rare *as a group*, constituting 1 to 5 per cent (average about 2 per cent) of all types of organic heart disease in the United States. Individual abnormalities are exceedingly rare, there being only a few which may be considered relatively common. It is impossible to discuss all of the anomalies which have been described in the medical literature; only the salient features of the most common ones will be discussed briefly. Many of the congenital defects are

not important clinically since some are not compatible with postnatal life Others are so mild as to have little influence on life expectancy or morbidity

The *cause* of congenital abnormalities remains conjectural Such factors as malnutrition x radiation virus infection during pregnancy, alcoholism drug addiction syphilis and age probably play a rôle in predisposing to disturbances in intrauterine development which leads to congenital defects Their mechanism of action or even whether they act etiologically in some ... ...tant to remember that the incidence ... cent if the mother has ... er of pregnancy This ... pregnancy So high is the incidence that abortion should be recommended if the mother has rubeola during the first three months of pregnancy The embryologic development of the heart and great vessels and the normal fetal and adult circulations should be reviewed by the student at this point

The incidence of the various defects is shown in Table 13 The detailed clinical classification of congenital anomalies of the heart by Abbott may be consulted Only the common defects are discussed

TABLE 13 – THE INCIDENCE OF THE MORE COMMON TYPES OF CONGENITAL HEART DISEASE (FROM ABBOTT'S 1000 PATIENTS)

	Per cent
Interatrial septal defect	37
Interventricular septal defects	25
Patent ductus arteriosus	24
Pulmonary stenosis	15
Anomalies of the semilunar valves	13
Coarctation of the aorta (adult type)	11
Anomalies of the great veins	[illegible]
Complete transposition of the arterial trunks	7

The *clinical manifestations* of congenital defects in general are the same as those manifestations previously described for the other etiologic types of heart disease Certain phases of the clinical picture are fairly characteristic and important and are especially significant in diagnosis

1 *Age* Congenital defects begin in *fetal life* and therefore with few exceptions manifest themselves at *birth* ...

... *examination* a ... is not frequently performed in early life For this reason it is frequently difficult *to date the* cardiac disturbance to the period of fetal development

2 *Cyanosis* is another important part of the clinical picture Cyanosis occurs when there is enough *reduced* hemoglobin in the ... to result ...

of shunts and other defects that allow reduced hemoglobin to flow to the left side of the heart (Fig 147) Shunts which allow oxygenated hemoglobin to flow into reduced hemoglobin on the right side of the heart will not produce cyanosis (Fig 147)

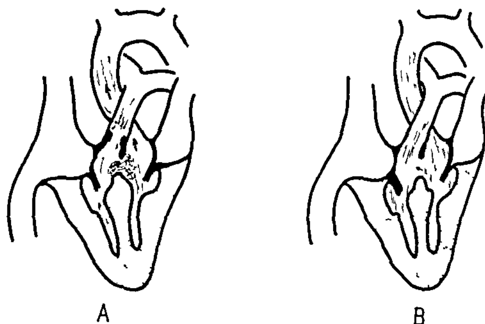


Fig 147 —Illustration showing how uncomplicated patent interventricular septal defect *B* results in escape of blood from the left ventricle with higher pressure into the right ventricle with lower pressure Since aerated blood enters the right side of the heart *no cyanosis* results In part A an interventricular septal defect is complicated by pulmonary stenosis When the stenosis is great enough to increase the pressure in the right ventricle above that in the left then unaerated blood escapes into the left side of the heart and flows into the general circulation The percentage reduced hemoglobin in the circulation may be increased enough to produce *cyanosis*

3 *Clubbing* The fingers and toes are clubbed in congenital heart disease if there is a long-standing disturbance in oxygenation of the blood or cyanosis

4 *Growth* Any type of heart disease which involves a disturbance with circulation to the tissues results in impairment in tissue nutrition Such alterations in metabolism when acquired in adult life do not manifest growth impairment, since growth has been completed Congenital heart disease however occurs early in life when growth and body development are rapid Impairment of the circulation and oxygen supply to the tissues retards or stops growth both mental and physical Such impairment is usually *diffuse* and the degree of impairment is more or less directly related to the degree of insufficiency in circulation and oxygen supply It may be limited to certain regions however, in some types of coarctation

5 *Murmurs* Congenital defects are somewhat peculiar and differ from acquired heart disease in that the murmurs tend to be highly developed and often are located outside the four clinical valvular areas, for

example patent ductus arteriosus and interventricular or interatrial septal defects. Some of these murmurs will be discussed more fully later.

6. *Electrocardiogram.* The electrocardiogram as a rule is not characteristic of congenital heart disease. There are individual exceptions to this rule. For example, the electrocardiographic pattern of situs inversus viscerum with dextrocardia is so characteristic that if properly recorded the anomaly can be detected by the electrocardiogram alone. Extreme right axis deviation is common and most highly developed in congenital heart disease.

7. *Secondary Polycythemia.* This is present in anomalies in which the arterial hemoglobin is relatively low in oxygen, resulting in a greater than normal rate of production of erythrocytes by the hemopoietic system. Other *blood chemical* changes, such as in CO_2, O_2 and pH, occur when oxygen-poor hemoglobin is delivered to the tissues.

8. *Cardiac Catheterization.* Most patients with congenital cardiac anomalies who live long enough to require clinical study for a detailed diagnosis and for possible surgical therapy usually can obtain an accurate, complete diagnosis without catheterization studies. When, however, diagnosis is difficult due to peculiarities in the clinical picture or to satisfy the patient and physician more definitely, cardiac catheterization may be conducted. It is important to remember that such studies offer danger, expense and psychic trauma to many people and therefore should be performed only when definitely indicated. Many more people are catheterized than is necessary.

Nevertheless, because catheterization is becoming more popular and because of increasing interest in cardiac surgery for treatment of congenital anomalies of the heart, it is advisable to review briefly the type of data obtainable by cardiac catheterization and to point out some of the principles employed by such studies. This is a highly specialized type of examination and, therefore, is developed fully in this presentation.

Cardiac catheterization is employed clinically to provide the following data:

a. Values of *pressure* in the great vessels of the heart and the cardiac chambers that can be entered by the catheter.

b. Values of the O_2 *content* and percentage O_2 saturation of the blood contained in the great vessels of the heart and in the c... [illegible]

c. [illegible] ...ac chambers is obtained either by roentgenographic recording of the position of the radiopaque catheter or by injection of radiopaque material or by both. Fluoroscopy, teleoroentgenography and/or cinefluoroscopy are employed in angiocardiography.

From these data it is possible to estimat... [illegible]

The average values of pressures and oxygen content of the blood in the great vessels and chambers of the normal child are shown diagrammatically by Figure 148 and in the normal adult by Figure 149 The conventional diagram is employed in all these illustrations The variations in these values and angiocardiographic data which occur from infancy to old age can be obtained from the literature The application of catheterization

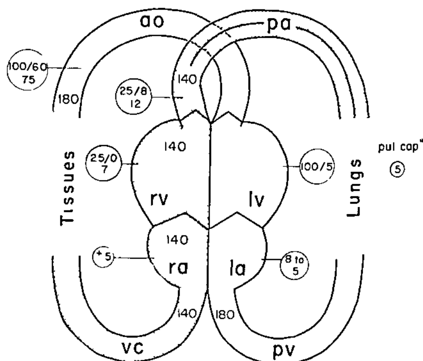
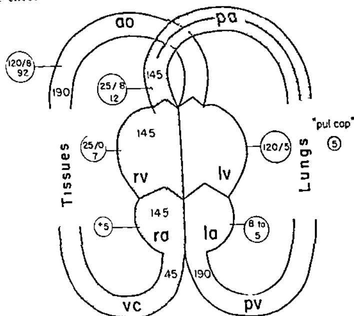


Fig 148 Diagrammatic representation of normal pressures and contents of oxygen in the blood of the heart and great vessels of the *normal child* In this illustration and all similar ones to follow the values enclosed in circles represent blood pressure, the mean values being shown below the respective systolic and diastolic values The values for oxygen content expressed in cubic centimeters of oxygen contained in one liter of whole blood are indicated within the respective cardiac chambers and vessels The value for pulmonary capillary pressure is the pressure obtained after firmly forcing
artery which is then arbitrarily called pulmonary
oxygen consumption is indicated at the bottom of

to the diagnosis of selected congenital defects is presented later It is possible from the discussion of these defects to learn the general principles of catheterization as applied to congenital cardiac anomalies

The methods and apparatus employed in catheterization studies are relatively crude and are subject to error from many sources The method for measuring the O_2 content in a sample of blood is sufficiently accurate for clinical purposes to offer no serious difficulties provided the technic is

acies of the apparatus itself. The c... poor physical characteristics as a tube for transmitting pressure to a pressure recorder outside the patient. New recorders with the pick up unit placed directly within the heart are being developed. For most clinical purposes the pressures recorded are usually sufficiently accurate for diagnostic purposes. The main difficulties are physiologic ones which occur within the patient himself and which are difficult to control. These include effects of anesthesia on the circulation, failure of the patient to



Oxygen consumption at rest - 185 cc./min.

Fig. 141. The pressures and contents of oxygen in the blood of the normal adult

relax during studies or to relax equally on repeated studies, and failure of blood to mix adequately within the great vessels and chambers of the heart. The latter offer particular difficulties. Because of the aforementioned problems the physician should never accept without considerable reservation absolute values of pressure, O_2 content or O_2 saturation of blood, calculations of the sizes of shunts, and the like. Only general trends of values and their correlation with the basic clinical data should be employed if good cardiologic diagnosis and management are to be achieved.

Cardiac catheterization performed by way of the venous system is relatively safe, whereas by way of the arteries it is dangerous and should be limited to *special problems only*.

The average values of pressures and oxygen content of the blood in the great vessels and chambers of the normal child are shown diagrammatically by Figure 148 and in the normal adult by Figure 149. The conventional diagram is employed in all these illustrations. The variations in these values and angiocardiographic data which occur from infancy to old age can be obtained from the literature. The application of catheterization

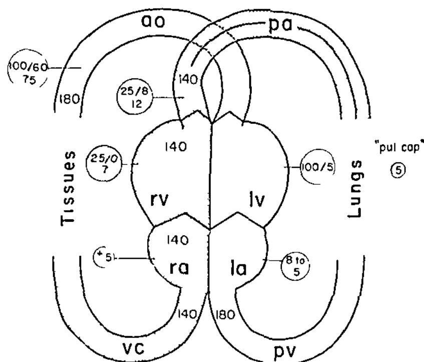


FIG 148 — Diagrammatic representation of normal pressures and contents of oxygen

for oxygen content, expressed in cubic centimeters of oxygen contained in one ... whole blood are indicated within the respective cardiac chambers and vessels. The value for "pulmonary capillary" pressure is the pressure obtained after firmly forcing the catheter into a small pulmonary artery which is then arbitrarily called "pulmonary capillary" pressure. *The rate of oxygen consumption is indicated* at the bottom of each such illustration.

to the diagnosis of selected congenital defects is presented later. It is
these defects to learn the general principles
congenital cardiac anomalies
employed in catheterization studies are
relatively crude and are subject to error from many sources. The method for measuring the O_2 *content in a sample of blood is sufficiently* accurate for clinical purposes, to offer no serious difficulties provided the technic is

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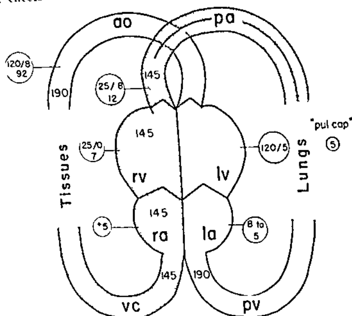


Fig. 110. . . pressures and contents of oxygen in the blood of the normal adult.

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Cardiac catheterization performed by way of the venous system is relatively safe, whereas by way of the arteries it is dangerous and should be limited to *special problems only*.

Anomalous Shunts

From data of O_2 content and consumption by the tissues it is possible not only to determine types of anomalous shunts but also to estimate the size of shunts in terms of blood flow through the openings. Beginners often have difficulty understanding these calculations due usually to failure to learn the simple principles involved. These principles of calculating shunts will be briefly reviewed. Certain simple formulae are employed in the calculations which can be no more accurate than the data employed in them. Remember that the data are often highly inaccurate as previously indicated. The excellent work of Cournand and his associates in the development of the method of cardiac catheterization is acknowledged. Most of the ideas and data presented below originate from them.

Symbols. *Oxygen concentration* in the sampled blood is expressed in cubic centimeters per liter of blood. This includes oxygen dissolved in the blood as well as that combined with hemoglobin.

C_{ao} = O_2 concentration in the aorta and peripheral arteries
C_{lv} = O_2 concentration in the left ventricle
C_{rv} = *O_2 concentration in the right ventricle*
C_{la} = O_2 concentration in the left atrium
C_{pv} = O_2 concentration in the pulmonary vein
C_{svc} = O_2 concentration in the superior vena cava
C_{ivc} = O_2 concentration in the inferior vena cava
$C_{\bar{v}} = \frac{C_{svc} + C_{ivc}}{2}$ This empiric value is subject to error if C_{svc} and C_{ivc} are not equal because more *blood flows through the inferior vena cava* than through the superior vena cava.
O_2 = Oxygen consumption in cubic centimeters per minute
BF_s = *Systemic blood flow in liters per minute*
BF_p = Pulmonary artery blood flow in liters per minute
BF_{pc} = Pulmonary capillary blood flow in liters per minute
SR = Shunt right to left in liters per minute
SL = Shunt left to right in liters per minute
SL = Shunt of blood from pulmonary veins or left atrium into right atrium
SL = *Shunt from left to right ventricle*
SL = Shunt from aorta into pulmonary artery
R = Regurgitated blood in liters per minute

Systemic blood flow is obtained from the equation

$$BF_s = \frac{O_2}{C_{ao} - C_{ra}} = \frac{O_2}{C_{ao} - C_{\bar{v}}} \qquad (1)$$

For example if a patient consumes 100 cc of O_2 per minute and the arterial O_2 concentration is 180 cc of O_2 per liter of blood and the right atrial blood concentration is 140 cc (Figure 150) then the systemic blood flow is

$$BF_s = \frac{100}{180 - 140} = 2.5 \text{ liters per minute}$$

Thus 100 cc of O_2 is being absorbed in the lungs per minute and if the patient is resting and is in a steady state he is using 100 cc per minute in the tissues If each liter of blood passing through the tissues delivers 40 cc (180 − 140) then $\frac{100}{40}$ or 2.5 liters must have circulated through the ... This is the ... represents ... If the state

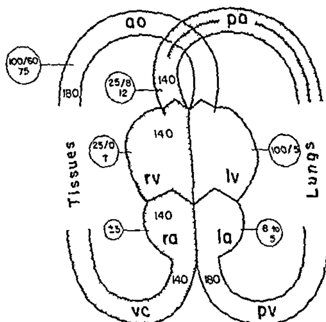


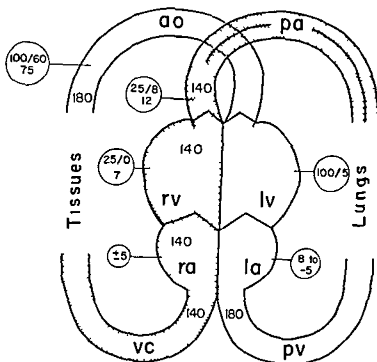
Fig 150 The pressures and contents of oxygen in the blood of a normal child obtained by cardiac catheterization Consult the text for the method of employing these values for calculation of the output of the left ventricle

of the circulation is normal then the output of the right ventricle is the same 2.5 liters ... e blood flow through ... ulmonary capillaries

$$\dot{m}_p = C_a - C_{pa} \quad (2)$$

For example, if the O_2 consumption is 100 cc per minute and the O_2 concentration in the left atrium is 180 and in the pulmonary artery 140 cc per liter (Figure 151), then

$$BF_p = \frac{100}{180 - 140} = 2.5 \text{ liters per minute}$$



Oxygen consumption at rest = 100 cc/min

[illegible] oxygen in the [illegible] of a normal child. [illegible] the text for the method of employing the [illegible] right ventricle

Thus, 100 cc of O_2 is added to the blood flowing through the lungs as determined by measurement of the O_2 consumed by the patient over a known period of time. Furthermore it is known that each liter of blood passing through the lungs contains 140 cc of O_2 and, after passage, it contains 180 cc of O_2, *i.e.*, each liter gathers 40 cc of O_2. But 100 cc of O_2 is gathered per minute, therefore $\frac{100}{40}$ or 2.5 liters must have circulated through the lungs to accomplish this (Figure 151).

Left to Right Intracardiac Shunt

a. **C_{ra} is greater than C_{vc}.** When the concentration of oxygen in the right atrial blood is greater than that in the vena caval blood, then the following defects may exist:

(1) Shunting of blood from the left atrium into the right atrium ... septal defect

(2)

(3)

through an interventricular septal defect ...
cuspid valvular regurgitation

(4) Any combination of the foregoing is possible.

The degree of left-to-right shunt may be determined from the equation

$$SI = BF_s\left(\frac{C_{ra} - C_{vc}}{C_{la} - C_{ra}}\right) \quad (3)$$

For example, if catheterization studies reveal data shown in Figure 152, then

$$SI = BF_s\left(\frac{100 - 60}{130 - 100}\right)$$

$$BF_s = \frac{40}{130 - 60} = 0.57 \text{ liter per minute}$$

$$SI = 0.57\left(\frac{100 - 60}{130 - 100}\right) = 0.57\left(\frac{40}{30}\right) = 0.76 \text{ liter per minute}$$

Thus it is evident from the calculation and Figure 152 that each liter of blood entering the right atrium from the left atrium contains 130 cc ... when mixed in the right ... per liter of blood. Note ... on of 60 cc. O_2 per liter. Therefore, to bring one liter of blood with 60 cc. concentration to 100 cc., 40 cc. O_2 must be added. Since each liter of blood from the left atrium adds 30 cc., it would require $\frac{40}{30}$ or 1.33 liters of the 130 cc. blood to do this. But the cardiac output is only 0.57 liter, i.e., only 0.57 liter of blood with 60 cc. per liter of O_2 is entering the right atrium per minute which is to be increased to 100 cc. O_2 saturation. Therefore it would require only 0.57 × 1.33 liter or 0.76 liters of the 130 cc. blood from the left atrium to accomplish the change.

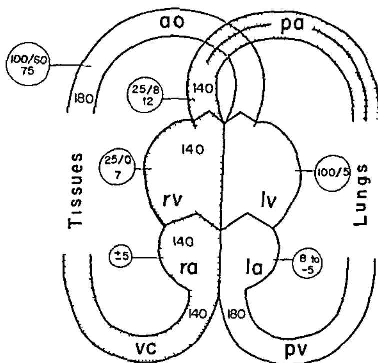
The degree of the shunt could also be obtained from a measurement of the difference between the cardiac outputs of the two ventricles.

$$BF_p = \frac{40}{130 - 100} = 1.33 \text{ liters}$$

$$SI = BF_p - BF_s = 1.33 - 0.57 = 0.76 \text{ liter per minute}$$

For example, if the O_2 consumption is 100 cc per minute and the O_2 concentration in the left atrium is 180 and in the pulmonary artery 140 cc per liter (Figure 151) then

$$BF_p = \frac{100}{180 - 140} = 2.5 \text{ liters per minute}$$



Oxygen consumption at rest = 100 cc/min

Fig. 151 The pressures and content of oxygen in the blood of a normal child obtained by cardiac catheterization. Consult the text for the method of employing these values for calculation of the output of the right ventricle

Thus, 100 cc of O_2 is added to the blood flowing through the lungs as determined by measurement of the O_2 consumed by the patient over a known period of time. Furthermore it is known that each liter of blood passing through the lungs contains 140 cc of O_2 and after passage it contains 180 cc of O_2, i.e. each liter gathers 40 cc of O_2. But 100 cc of O_2 is gathered per minute therefore $\frac{100}{40}$ or 2.5 liters must have circulated through the lungs to accomplish this (Figure 151)

Left to Right Intracardiac Shunt

— W... concentration of oxygen in the right

through an interatrial septum
(2) Shunting of blood from an aberrant pulmonary vein which empties into the right atrium
(3) Shunting of blood from the left ventricle into the right ventricle ... association with tri

(4)
The degree of left-to-right shunt may be determined from the equation

$$SI = BF_s\left(\frac{C_{ra} - C_{vc}}{C_{la} - C_{ra}}\right) \quad (3)$$

For example if catheterization studies reveal data shown in Figure 152 then

$$SI = BF_s\left(\frac{100 - 60}{130 - 100}\right)$$

$$BF_s = \frac{40}{130 - 60} = 0.57 \text{ liter per minute}$$

$$SI = 0.57\left(\frac{100 - 60}{130 - 100}\right) = 0.57\left(\frac{40}{30}\right) = 0.76 \text{ liter per minute}$$

Thus, it is evident from the calculation and Figure 152 that each liter of blood entering the right atrium from the left atrium contains 130 cc O_2. It is reduced to 100 cc O_2 saturation per liter when mixed in the right atrium that is there is a decline or loss of 30 cc per liter of blood. Note that it is mixing with blood with a concentration of 60 cc O_2 per liter. Therefore ... 60 cc concentration to 100 cc
40 ... of blood from the left atrium

... would require $\frac{40}{30}$ or 1.33 liters of the 130 cc blood to do this.

But the cardiac output is only 0.57 liter i.e. only 0.57 liter of blood with 60 cc per liter of O_2 is entering the right atrium per minute which is to be increased to 100 cc O_2 saturation. Therefore it would require only 0.57 × 1.33 liter or 0.76 liters of the 130 cc blood from the left atrium to accomplish the change.

The degree of the shunt could also be obtained from a measurement of the difference between the cardiac outputs of the two ventricles

$$BF_p = \frac{40}{130 - 100} = 1.33 \text{ liters}$$

$$SI = BF_p - BF_s = 1.33 - 0.57 = 0.76 \text{ liter per minute}$$

b C_{rv} is greater than C_{ra} For this to occur blood must be shunting into the right ventricle from the left ventricle mixing with the blood of lower O_2 concentration entering the right ventricle from the right atrium The size of the shunt is calculated from the equation

$$SF' = BF_s\left(\frac{C_{rv} - C_{ra}}{C_v - C_{rv}}\right) \tag{4}$$

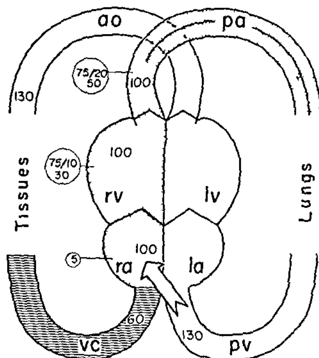


Fig 152 —Pressures and contents of oxygen in the blood of a child with interventricular septal defect

For example from data obtained by cardiac catheterization and summarized by Figure 153 the amount of blood shunting through the interventricular defect is

$$SF' = 3.4\left(\frac{120 - 110}{135 - 120}\right) = 2.24 \text{ liters per minute}$$

$$\text{where } BF_s = \frac{85}{135 - 110} = 3.4 \text{ liters}$$

Thus from equation (4) if blood from the left ventricle containing 135 cc O_2 per liter mixes with one liter of blood from the right ventricle con

taining 110 cc O_2 per liter to produce blood containing 120 cc O_2 per liter then the 135 cc O_2 per liter blood would be reduced 15 cc for each liter of right ventricular blood raised from 110 to 120 cc O_2 per liter, that is enough blood from the left ventricle must enter the right ventricle to add 15 cc O_2 to each liter of blood in the right ventricle. Thus $\frac{10}{15} \times 1$ or 0.66 liter of blood must be added to each liter of the 110 cc O_2 blood in the left ventricle. But 3.4 liters of blood flows through the right ventricle per minute, therefore, 0.66×3.4 or 2.2 liters must shunt from the left ventricle into the right one.

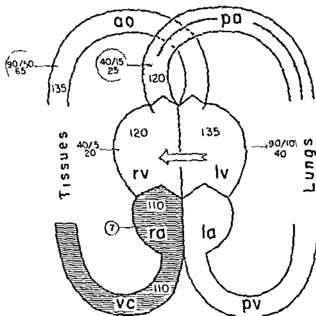


Fig. 153. Pressures and contents of oxygen in the blood of a child with a ventricular septal defect

The volume of the shunt may also be calculated from the difference between the right and left ventricular outputs, that is

$$SI = BF_p - BF_s = 5.6 - 3.4 = 2.2 \text{ liters per minute}$$

(c) **C_{rv} is greater than C_{ra} and C_{ra} is greater than C_v.** For the O_2 content of blood from the right ventricle (C_{ra}) to be greater than that of blood from the venae cavae (C_{vc}) oxygenated blood must shunt into the right ventricle from the left atrium or an aberrant pulmonary vein. Further-

more, for the O_2 content of the blood in the right ventricle (C_{rv}) to be greater than that in the right atrium (C_{ra}) blood must also shunt from the left side of the heart, left ventricle into the right side right ventricle. Therefore, there must be two left-to-right shunts. An interventricular septal defect with tricuspid regurgitation could produce the foregoing conditions.

$$SI = (BI + SI)\left(\frac{C_{rv} - C_{ra}}{C_{lv} - C_{rv}}\right) \qquad \text{([illegible])}$$

For example, from the data shown in Figure 154, the amount of blood shunting through an interventricular shunt is obtained as follows:

Oxygen consumption at rest 85 cc/min

Fig. 154.—Pressures and contents of oxygen in the blood of a child with both atrial and ventricular septal defects.

$$BI_s = \frac{85}{135 - 110} = 3.4 \text{ liters per minute}$$

$$SI' = BI_s\left(\frac{C_{ra} - C_{vc}}{C_{la} - C_{ra}}\right) = 3.4\left(\frac{115 - 110}{135 - 115}\right) = 0.85 \text{ liters per minute}$$

Then

$$SL = (34 + 0.85)\left(\frac{C_{rv} - C_{ra}}{C_{lr} - C_{rv}}\right) =$$

$$4.25\left(\frac{120 - 115}{135 - 120}\right) = 1.4 \text{ liters per minute}$$

The explanation of the mathematics is as follows. Blood from the venae cavae containing 110 cc O_2 per liter enters the right atrium. To this blood is added blood from the left atrium which increases its O_2 content to 115 cc per liter. Therefore, from this it is possible to calculate the left-to-right shunt at the atrial level (SL) by the same reasoning applied to equation (3). Once the volume of this shunt is known and the systemic blood (BF_s) is obtained from equation (1), it is possible to know the volume of blood that enters the right ventricle. This is $BF_s + SL$. Furthermore, it is known that blood with 115 cc O_2 per liter enters the right ventricle, and its O_2 content is raised to 120 cc per liter. From these data it is then po... [illegible]

the size of combinations of shunts.

d. **C_{pa} is greater than C_{rv} and C_{rv} is greater than C_{ra}.** The condition in which the O_2 concentration of the blood is (1) greater in the pulmonary artery than in the right ventricle and (2) greater in the right ventricle than in the right atrium is usually due to

(1) C_{pa} is greater than C_{rv} due to shunting of blood from the aorta into the pulmonary artery because of either (a) patent ductus arteriosus or (b) patency of the septum between the pulmonary artery and the aorta. This permits oxygenated blood to enter the pulmonary artery and cause C_{pa} to exceed C_{rv} and

(2) C_{rv} is greater than C_{ra} due to either (a) shunting of blood from the left ventricle through a patency in the interventricular septum into the right ventricle, (b) pulmonary valvular insufficiency with regurgitation of the more highly oxygenated blood from the pulmonary artery into the right ventricle, or (c) both these conditions.

The volume of the shunt from the aorta into the pulmonary artery is obtained from the equation

$$SL = (BF_s + SL)\left(\frac{C_{pa} - C_{rv}}{C_{ao} - C_{pa}}\right) \qquad (6)$$

For example, from P... [illegible]

As in the preceding illustration and in the solution of equation (5), this equation must be approached in steps and in the proper order, according to the same line of reasoning as was applied to the previous equations and solutions.

For example, if the condition shown in Figure 155 is to be solved for the shunting through the patent ductus arteriosus, the solution would be

$$SF' = \left[\left(\frac{O_2}{C_{ao} - C_{ra}}\right) + \left(\frac{O_2}{C_{ao} - C_{ra}}\right)\left(\frac{C_{rv} - C_{ra}}{C_{lv} - C_{rv}}\right)\right]\left(\frac{C_{pa} - C_{rv}}{C_{ao} - C_{pa}}\right) \qquad (7)$$

$$= \left[\left(\frac{100}{150 - 100}\right) + \left(\frac{100}{150 - 100}\right)\left(\frac{110 - 100}{150 - 110}\right)\right]\left(\frac{115 - 110}{150 - 115}\right)$$

$$= [2 + 2(0.25)]\,0.14 = 2.5 \times 0.14 = 0.35 \text{ liters per minute}$$

The interventricular shunt is 0.5 liter per minute, which is obtained from the second fraction in equation (7).

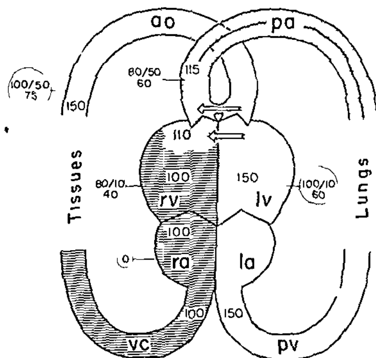


FIG. 155.—Pressures and contents of oxygen in the blood of a child with both ventricular septal defect and patent ductus arteriosus.

c. If the O content of the blood in the right ventricle were due to regurgitation of blood through an incompetent pulmonary valve, then the size of the regurgitation R could be derived from the equation

$$R = BF_s\left(\frac{C_{rv} - C_{ra}}{C_{pa} - C_{rv}}\right) \qquad (8)$$

Then for the example above there is patent ductus arteriosus and no ventricular septal defect but pulmonic valvular insufficiency (Figure 136) the degree of the insufficiency being

$$R = 2\left(\frac{110 - 100}{115 - 110}\right) = 2 \times 2 = 4 \text{ liters per minute}$$

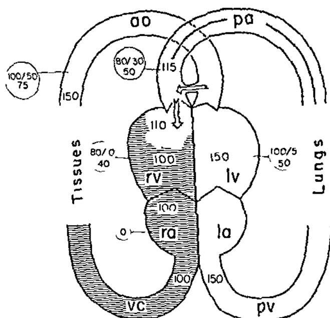


Fig. 136. Pressures and contents of oxygen in the blood of a child with both patent ductus arteriosus and pulmonic valvular incompetency

Right to Left Intracardiac Shunt

When this occurs the oxygen content of the blood which should be adequate is low. This type of shunting could occur by the passage of blood from the right atrium through an [illegible] left atrium or there [illegible] or anatomic overri[illegible] seen with tetralogy [illegible] degree of right-to-left shunting of blood can be calculated by the equation

$$SR = BL_a\left(\frac{C_{la} - C_{ao}}{C_{la} - C_{vc}}\right) \tag{9}$$

For example, if the values shown in Figure 157 are employed, the degree of right to left shunt, *i.e.*, the amount of blood which is shunted directly into the left side of the heart in the case of tetralogy of Fallot is

$$SR' = \frac{110}{85 - 30}\left(\frac{200 - 85}{200 - 30}\right) = 1.4 \text{ liters per minute}$$

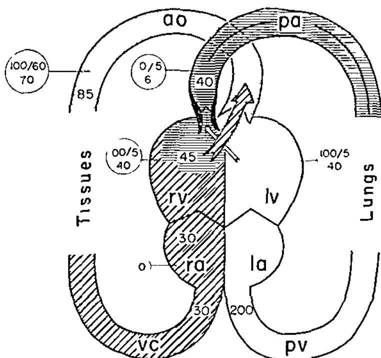


Fig. 157.—Pressures and contents of oxygen in the blood of a child with tetralogy of Fallot.

This is evident from the following argument. The oxygen in the blood shunted through the ventricular defect into the aorta is SR (30); the oxygen entering the aorta from the left ventricle is $(BF_s - SR)\,200$, and therefore the oxygen entering the aorta from both ventricles is $(BF_s - SR + SR)\,85$.

Thus

$$85\,(BF_s - SR + SR) = 30\,SR + 200\,(BF_s - SR)$$

$$SR\,(200 - 30) = BF_s\,(200 - 85)$$

$$\text{or } SR = BF_s\left(\frac{200 - 85}{200 - 30}\right)$$

That the preceding calculation is correct is evident from the following argument. In all of these studies it is assumed that the oxygen used in the tissue per minute is equal to the oxygen absorbed from the lungs per minute, i.e.

$$\text{Oxygen from lungs} = \text{oxygen to tissues} \qquad (a)$$

The volume of O_2 delivered to the tissues per minute is $BF_s(C_{ao} - C_{vc})$ and the oxygen absorbed from the lungs is $(BF_s - SR')(C_{1s} - C_{pa})$ where the volume of blood flow to the lungs is equal to the systemic blood flow minus the volume of the shunt. Therefore, substituting in equation (a)

$$BF_s(C_{ao} - C_{vc}) = (BF_s - SR')(C_{1s} - C_{pa}) \qquad (b)$$

By multiplying and changing signs

$$-BF_s(C_{ao} - C_{vc}) + BF_s(C_{1s} - C_{pa}) = SR'(C_{1s} - C_{pa})$$

$$SR' = BF_s\left(\frac{C_{1s} + C_{vc} - C_{ao} - C_{pa}}{C_{1s} - C_{pa}}\right) \qquad (c)$$

Since $C_{vc} = C_{pa}$ then

$$SR' = BF_s\left(\frac{C_{1s} - C_{ao}}{C_{1s} - C_{vc}}\right) \qquad (d)$$

Thus it is evident that the general equation is correct.

It is not possible nor necessary in this presentation to discuss in detail the methods for calculating shunts through congenital defects. Other equations have been advanced and more complex multiple defects can exist which are available in the medical literature.

Specific Cardiac Defects

Certain of the more common congenital cardiac lesions will be discussed primarily from the point of view of the clinical manifestations at the bedside. Typical findings on cardiac catheterizations will be briefly summarized indicating the changes in (1) *pressure* relationships and (2) O_2 content of the blood. The *degree of shunting* will be ignored since absolute values are not presented.

The *incidence* of congenital heart disease may be estimated from the study of 200 patients by Paul Wood (Table 14). Roughly tetralogy of Fallot and atrial septal defect each represent about 20 per cent or one-fifth of the congenital cardiac anomalies. The incidence of patent ductus arteriosus is about 15 per cent. Ventric[illegible]

Four fifths

applications

TABLE 14.—INCIDENCE OF TRUE CONGENITAL HEART DISEASE

Type of Lesion	*Per cent*
Acyanotic	
Coarctation of the aorta	8
Aortic or subaortic stenosis	3
Simple pulmonary stenosis (4 with V.S.D.)	11.5
Atrial septal defect	17.5
Ventricular septal defect (excluding 4 with pulmonary stenosis)	12
Patent ductus arteriosus	14.5
Cyanotic	
Fallot's tetralogy (including 3 with pulmonary atresia)	18
Pulmonary stenosis with reversed interatrial shunt	2.5
Tricuspid atresia	3
Eisenmenger's complex	1
Transposition of the great vessels	1
Others	8
TOTAL	100

Modified from Wood, Paul: Brit. Med. J. 2: 540, 1950.

TABLE 15.—CLASSIFICATION OF CONGENITAL HEART DISEASE

No Shunt

General
- Dextrocardia
- Idiopathic hypertrophy
- Von Gierke's disease
- Heart block
- Familial cardiomegaly

Left-sided
- Coarctation of the aorta
- Right-sided aortic arch
- Complete or incomplete aortic rings
- Bicuspid aortic valve (or supernumerary cusps)
- Aortic or subaortic stenosis
- Left coronary artery arising from pulmonary artery

Right-sided
- Idiopathic dilatation of the pulmonary artery
- Simple pulmonary stenosis (with or without patent F.O.)
- Ebstein's disease

With Shunt

Acyanotic

Left-to-right Shunt (Pulmonary Plethora)
- Left ventricular enlargement
  - Patent ductus
  - Ventricular septal defect (with or without mild pulmonary stenosis)
  - Perforated aortic sinus into P.A. or R.V. (or R.A.)
- Right ventricular enlargement
  - Atrial septal defect (with or without mild pulmonary stenosis)
  - Anomalous pulmonary veins joining S.V.C. or R.A.

Cyanotic

Right-to-left Shunt
- Diminished pulmonary blood flow
  - Low P.A. pressure
    1. Left ventricular enlargement
       - Tricuspid atresia
    2. Right ventricular hypertrophy
       - Fallot's tetralogy
       - Pulmonary atresia (Fallot type)
       - Persistent truncus
       - Pulmonary stenosis with reversed interatrial shunt
  - High P.A. pressure
    - Eisenmenger's complex
    - Pulmonary hypertension with reversed aorto-pulmonary, interventricular, or interatrial shunt
- Pulmonary plethora
  - Transposition

Modified from Wood, Paul: Congenital Heart Disease. Brit. Med. J. [illegible] 1950.

Auricular Septal Defects

One of the most frequent of all cardiac anomalies is patency of the interauricular septum which occurs in about 20 per cent of all congenital cardiac anomalies The most frequent type is *patency of the foramen ovale* A small opening of the foramen is found in 12 to 25 per cent of all autopsies In many instances the opening is too small to produce an audible murmur or sufficient disturbances in hemodynamics to reveal even the slightest clinical evidence of the defect From a practical clinical point of view this is of no importance

An *auricular septal defect* on the other hand, is of clinical importance, since it produces at least a murmur and in many instances when large, considerable disturbance in hemodynamics In this type of *defect an*

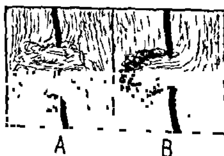


Fig 158 —Diagram A shows how the blood from both sides of the ...

... type of defect obviously does not produce cyanosis

al normally large amount of blood enters the right atrium from the left This increases the load on the right ventricle Blood flows from the left atrium to the right because the pressure is greater in the left atrium The fact that the left atrium is above or superior to the right one may account in part for this difference in pressure In the average patient with a relatively small opening there is no cyanosis as oxygenated blood flows into the right side along with reduced hemoglobin When the ... large (cor ...
mixing and ...
by large op...

There is a loud systolic *murmur* over the upper ...
level of ...
dur...
...al ...

The peripheral pulse is normal or small ... the left infraclavicular region and the precordium seems to lift or move forward with the heart beat This is due to the *right ventricular enlargement* especially of the outflow tract With enlargement of the left ventricle, the precordium seems to

Type of Lesion	Per cent
Acyanotic	
Coarctation of the aorta	8
Aortic or subaortic stenosis	3
Simple pulmonary stenosis (1 with V. S. D.)	11.5
Atrial septal defect	17.5
Ventricular septal defect (excluding 1 with pulmonary stenosis)	12
Patent ductus arteriosus	14.5
Cyanotic	
Fallot's tetralogy (including 3 with pulmonary atresia)	18
Pulmonary stenosis with reversed interatrial shunt	2.5
Tricuspid atresia	3
Eisenmenger's complex	1
Transposition of the great vessels	1
Others	8
TOTAL	100

Modified from Wood, Paul: Brit. Med. J. *2*: 540, 1950.

TABLE 15.—CLASSIFICATION OF CONGENITAL HEART DISEASE

No Shunt

General	*Left-sided*	*Right-sided*
Dextrocardia	Coarctation of the aorta	Idiopathic dilatation of the pulmonary artery
Idiopathic hypertrophy	Right-sided aortic arch	*Simple pulmonary stenosis* (with or without patent F. O.)
Von Gierke's disease	*Complete or incomplete* aortic rings	Ebstein's disease
Heart block	Bicuspid aortic valve (or supernumerary cusps)	
Familial cardiomegaly	Aortic or subaortic stenosis	
	Left coronary artery arising from pulmonary artery	

With Shunt

Acyanotic

Left-to-right Shunt (Pulmonary Plethora)

- Left ventricular enlargement
  - Patent ductus
  - Ventricular septal defect (with or without mild pulmonary stenosis)
  - Perforated aortic sinus into P. A. or R. V. (or R. A.)
- Right ventricular enlargement
  - Atrial septal defect (with or without mild pulmonary stenosis)
  - Anomalous pulmonary veins joining S. V. C. or R. A.

Cyanotic

Right-to-left Shunt

- Diminished pulmonary blood flow
  - Low P. A. pressure
    1. Left ventricular enlargement
       - Tricuspid atresia
    2. Right ventricular hypertrophy
       - Fallot's tetralogy
       - Pulmonary atresia (Fallot type)
       - Persistent truncus
       - Pulmonary stenosis with reversed interatrial shunt
  - High P. A. pressure
    - Eisenmenger's complex
    - Pulmonary hypertension with reversed aorto-pulmonary interventricular or interatrial shunt
- *Pulmonary plethora*
  - Transposition

Modified from Wood, Paul: Congenital Heart Disease. Brit. Med. J. 2: 698, 1950.

TABLE 17.—CYANOTIC

Diagnosis	Lung Fields	Size and Shape of Heart	Pulmonary Window	Pulmonary Segment	Electrical Preponderance
Pulmonary Flow Less than Systemic Flow. Pulmonary Artery Pressure Usually Decreased					
Tetralogy of Fallot	Clear	Elevated apex not enlarged "coeur en sabot"	Clear	Concave	Right
Pseudotruncus	Clear	Enlarged and boot shaped	Clear	Concave	Right
Tricuspid Atresia	Clear	Concavity of lower right border in P A projection "coeur en sabot"	Clear	Concave	Left
Transportation of the Great Vessels with Pulmonary Stenosis	Clear	Enlarged, narrow mediastinal shadow in A P and lateral positions	Hazy	Concave	Combined Heart Strain
Ebstein's Disease with Patent Foramen Ovale	Clear	Enlarged	Hazy	Convex	Right or Left with bundle branch block
Pulmonary Arterio-Venous Fistula	Round Opacities	Enlarged or normal	Hazy	Convex	Right
Patent Foramen Ovale or Auricular Septal Defect with Pulmonary Stenosis	Clear	Slightly enlarged	Hazy	Convex	Right
Pulmonary Flow Greater than Systemic Flow and or Pulmonary Artery Pressure Normal or Increased					
Eisenmenger's	Vascular	Enlarged	Hazy	Convex	Right
Complete Transposition	Vascular	Enlarged, narrow mediastinal shadow in A P, widening in lateral view, absence of aortic knob	Hazy		Right
Patent Ductus Arteriosus with Reversed Flow	Vascular	Enlarged	Hazy	Convex	Right
Truncus Arteriosus	Vascular	Enlarged and boot shaped	Clear	Concave	Left or right

The "Lung Fields", "Size and Shape of Heart", "Pulmonary Window" and "Pulmonary Segment" columns are grouped under *Fluoroscopic Findings*.

Courtesy of Dr Richard Bing

TABLE 16 — NON CYANOTIC

Pulmonary Flow Greater than Systemic Flow and/or Pulmonary Artery Pressure Normal or Increased

Diagnosis	*Fluoroscopic Findings*				*Electrical Preponderance*
	Lung Fields	*Size and Shape of Heart*	*Pulmonary Window*	*Pulmonary Segment*	
Isolated Septal Defect Auricular					
Uncomplicated	Vascular	Enlarged	Hazy	Convex	Right
Lutembacher	Vascular	Enlarged	Hazy	Very Prominent	Right
Ventricular Septal Defect	Vascular	Usually enlarged	Hazy	Convex	Left or right
Patent Ductus Arteriosus	Vascular	Enlarged	Hazy	Convex	Normal or Left
Aortic Septal Defect	Vascular	Enlarged	Hazy	Convex	Normal or Left
Anomalous Venous Return with Pulmonary Vein Emptying into Vena Cava or Right Auricle	Vascular	Enlarged	Hazy	Convex	Right
Pulmonary Flow Equals Systemic Flow at Rest and After Exercise					
Pure Pulmonic Stenosis	Clear	Enlarged	Hazy	Prominent	Right

Courtesy of Dr Richard Bing

c The *left ventricular* impulse is increased and there is lifting of the precordium produced by the enlarged *right ventricular* outflow tract

d Pulsation of the *pulmonary artery* is felt

e A *systolic murmur* (Roger s murmur) most intense just to the left of the sternum at the level of the fourth intercostal space (Roger s area) is fairly characteristic A *thrill* is usually associated with the murmur

f The *pulmonary second sound* is split with the second or pulmonary portion accentuated

g A *functional mitral diastolic murmur* may be present *Functional pulmonary valvular insufficiency* with a pulmonary diastolic murmur may be noted

4 The *roentgenographic studies* reveal a large pulmonary conus large left ventricle and pulmonary plethora The findings are essentially the same as for patent ductus arteriosus

5 The *electrocardiogram* may show evidence of right ventricular enlargement Right bundle branch block complete or incomplete may be present With small defects the electrocardiogram may be normal

6 *Cardiac catheterization* reveals an increase in O_2 content of the blood found in the right ventricle and pulmonary artery The pressure in this ventricular and pulmonary artery is increased The shunt can be calculated as *previously indicated* The catheter may be passed into the left ventricle, which definitely establishes the abnormal opening

7 *Angiocardiography* does not show the patent septum because blood is flowing from left to right

8 Life is *shortened* if the opening is large but is not necessarily affected by a small opening

Patent Ductus Arteriosus

Surgical intervention has made this defect *reversible*

1 *Incidence* It represents about 15 per cent of all congenital anomalies It occurs about two to three times more often in women than in men although the reason for the difference by sex and the mechanism for normal closure of the ductus are not yet clearly understood

2 The size of the opening of the duct varies from less than 1 mm to 10 mm or more in diameter There may be a direct communication between the pulmonary artery and aorta or the d[illegible] by several centimeters [illegible] tus may be than a[illegible] pulmonary artery [illegible] About 30 per cen[illegible] infection *subacute bacterial endarteritis* at the site of the ductus Embolic phenomena may develop in both the pulmonary and systemic circulations being most pronounced in the lungs

There are no *symptoms* unless the opening is large when the degree of shunting will result in impairment of systemic circulation and stunting of growth The symptoms of congestive heart failure may develop

"rock" and not to lift The cardiac impulse appears to tap The pulmonary artery pulsation is palpable The pulmonary second sound is well split Normally, the pulmonary second sound is split particularly in children The first part of the split sound originates from the aortic valve and the second one from the pulmonic Only a single sound is heard over the aortic and mitral areas and in the neck This sound originates from the aortic valve With pulmonary hypertension such as occurs with auricular septal defect, there is associated an accentuated second part of the split pulmonic second sound A split P_2 indicates that both the *aorta and pulmonary artery* are fully developed Wide splitting occurs if there is right bundle branch block The right bundle branch block pattern is common with auricular septal defect

The various roentgenographic studies reveal a prominence of the pulmonary conus large main pulmonary artery and its branches and all pulmonary vascular markings are increased The pulmonary vessels pulsate strongly and a hilar dance may be observed The left and right atria especially the latter are usually large

Catheterization studies reveal an increase in *pressure* in the right atrium and right ventricle and the *oxygen content* of the blood in the right atrium right ventricle and pulmonary artery is higher than normal The catheter is often passed through the auricular septal defect into the left atrium and pulmonary veins where fully oxygenated blood is obtained When this occurs the defect is definitely established The amount of blood shunted through the defect can be calculated by the method previously described

Angiocardiography is of little assistance in demonstrating the septal defect because the blood is shunted from left to right Its greatest value is found in eliminating other defects

Paradoxical embolism may occur that is an embolus from a thrombus on the right or venous side of the systemic circulation which passes through the septal opening and occludes an artery of the systemic circulation rather than a pulmonary artery

Associated *congenital* (said to be acquired by some observers) *mitral stenosis and interauricular septal defect* is called the Lutembacher syndrome The combined effects of the two defects are present The septal opening probably *prolongs life by relieving the strain imposed on the pulmonary* veins and left atrium by mitral stenosis

Ventricular Septal Defect

1 *Incidence* Ventricular septal defect constitutes about 12 per cent of the congenital defects encountered clinically

2 *Symptoms* are usually absent unless there is a large opening

3 *Signs*

a There is *no cyanosis* unless a large septal opening with churning and mixing of blood exists (Fig 108)

b The *peripheral pulse is small or normal*

lances and their manifestations are the same as those found in aortic insufficiency and the same peripheral vascular signs (capillary pulsations, Traube's pistol shot sign, Corrigan pulse, etc.) may be present.

4. The *roentgenographic studies* are important. They reveal enlargement of the pulmonary conus, main pulmonary artery and its main branches. There is an increase in the pulmonary vascular markings with ... There may be a hilar dance

... It shows left ventricular hypertrophy which varies directly in degree with the size of the ductus opening. There may be evidence of right ventricular hypertrophy in some instances.

6. *Cardiac catheterization* reveals an increase in the O_2 content of the blood obtained from the pulmonary artery. The pulmonary arterial pressure is increased. A catheter passed through the patent ductus into the aorta will definitely establish the abnormal communication.

7. *Angiocardiography* is usually of little assistance since this is a left to right shunt, although the radiopaque material may be noted to be reduced in concentration at the point of entrance and distal to its entrance in the pulmonary artery. Its greatest value is in eliminating or detecting other lesions.

8. The *course* of the disease is variable. If the ductus opening is small, life expectancy is not altered by the defect, but if large, congestive heart failure and impaired growth develop. Subacute bacterial endarteritis occurs in about 30 per cent of patients. The offending organism is usually *Streptococcus viridans*.

9. *Treatment* consists in surgical ligation if the ductus is producing cardiac enlargement and impairment of function. With skilled surgical technic the mortality rate is less than 5 per cent, but with poorer technic it may approach 30 per cent. Subacute bacterial end...

... The cardiac state then slowly returns to normal and growth occurs rapidly.

Pulmonary Stenosis

A. Pulmonary Valvular Stenosis with Intact Atrial and Ventricular Septa

Uncomplicated pulmonary stenosis constitutes about 10 per cent of the congenital defects seen clinically.

1. *Incidence* is about equal in both sexes.

2. The *degree* of stenosis varies. The clinical manifestations vary directly with the severity of the stenosis.

The *symptoms* ...

impairment of p...

3 *Signs*

a The *machine murmur* is diagnostic It is continuous with systolic *accentuation*, the normally split pulmonary second sound the second or pulmonary portion being accentuated (Fig 159) There may be but rarely is only a systolic murmur A *functional* mitral diastolic rumble may be present There is an associated *thrill* The murmur is heard best in the first or second intercostal space near the sternum

b There is no *cyanosis*, as oxygenated blood is shunted into the pulmonary artery, nor is there *clubbing* of the fingers and toes

c The *blood pressure* shows a high pulse pressure due to high systolic pressure and low diastolic pressure The hemodynamic distur-

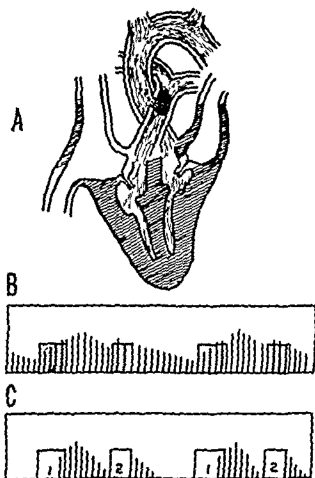


Fig 159 — Diagram illustrating patent ductus arteriosus and turbulence of blood flow from the aorta into the pulmonary artery The first diagram of the heart sounds and murmurs shows the characteristic continuous murmur with systolic exacerbations and accentuated P_2 *The second diagram shows the less characteristic but fairly common distinct systolic and diastolic murmurs* These are heard best over the P area *Consult the text for details*

bruits and their manifestations are the same as those found in aortic insufficiency and the same peripheral vascular signs (capillary pulsations, Traube's pistol shot sign, Corrigan pulse, etc.) may be present

4 The *roentgenographic studies* are important They reveal enlargement of the pulmonary conus main pulmonary artery and its main branches There is an increase in the pulmonary vascular markings with increased pulsations and pulmonary plethora There may be a hilar dance The right and left ventricles may be large

5 The *electrocardiogram* is not diagnostic It shows left ventricular hypertrophy which varies directly in degree with the size of the ductus opening There may be evidence of right ventricular hypertrophy in some instances

6 *Cardiac catheterization* reveals an increase in the O_2 content of the blood obtained from the pulmonary artery The pulmonary arterial pressure is increased A catheter passed through the patent ductus into the aorta will definitely establish the abnormal communication

7 *Angiocardiography* is usually of little assistance since this is a left to right shunt although the radiopaque material may be noted to be reduced in concentration at the point of entrance and distal to its entrance in the pulmonary artery Its greatest value is in eliminating or detecting other lesions

8 The *course* of the disease is variable If the ductus opening is small life expectancy is not altered by the defect but if large congestive heart failure and impaired growth develop Subacute bacterial endarteritis occurs in about 30 per cent of patients The offending organism is usually *Streptococcus viridans*

9 *Treatment* consists in surgical ligation if the ductus is producing cardiac enlargement and impairment of function With skilled surgical technic the mortality rate is less than 5 per cent but with poorer tech... it m... ...

... ... cured The cardiac state then slowly returns to normal, and growth occurs rapidly

Pulmonary Stenosis

A Pulmonary Valvular Stenosis with Intact Atrial and Ventricular Septa

Uncomplicated pulmonary steno... ... 10 per cent of the c... gen...

1

2

signs are primarily those of congestive heart failure. With exertion dyspnea may be severe. Squatting and substernal oppression or pain may be present and syncope may occur.

3 *Signs*

a *Cyanosis* is usually not present unless the stenosis is severe. It is peripheral in origin due to impairment of blood flow through the lungs.

b The patient may have a *bloated appearance* or a "moon" face.

c The *peripheral arterial pulse* tends to be small and weak.

d A *large a wave* in the jugular pulse is almost always present and may be noted with the naked eye.

e The *right ventricle* is felt to lift the precordium or to cause it to heave. The precordium bulges anteriorly with right ventricular enlargement.

f The *pulmonary* arterial impulse in the second intercostal space near the sternum is not felt because pulmonary arterial pressure is reduced.

g A harsh and widely distributed *systolic murmur* is always present in the pulmonary area. A *thrill* is usually associated with the murmur and may be present in the third intercostal space.

h The *pulmonary second heart sound* may be split, or there may be a single sound due to the presence of only the aortic component and absence of the pulmonic one, but it is not necessary that only a single sound be present.

4 *Roentgenographic* studies may reveal poststenotic dilatation of the main pulmonary artery. The pulmonary vascular markings are reduced and the pulmonary fields appear clear. There is right ventricular enlargement and the right atrium may be enlarged. The aorta appears to be small because of the low cardiac output. If there is mild pulmonary stenosis the pulmonary vascular markings will appear normal.

5 The *electrocardiogram* shows evidence of right ventricular hypertrophy. The pattern for complete or incomplete right bundle branch block may be present.

6 *Cardiac catheterization* reveals an increase in right atrial pressure and a definite increase in left ventricular pressure. The pressure in the pulmonary artery is greatly reduced. When the catheter is retracted from the pulmonary artery into the right ventricle while the pressure is continuously being recorded, a characteristic pressure record is obtained. The pressure suddenly rises from the low pressure distal to the stenosis recorded in the pulmonary artery to the high systolic pressure recorded in the right ventricle proximal to the stenosis. The pressure rises suddenly when the catheter tip is retracted from the pulmonary artery into the right ventricle proximal to the stenotic point.

7 *Angiocardiography* reveals delayed filling and visualization of the pulmonary arteries and in turn the left atrium and left ventricle. The

contrast medium is retained for a relatively long period of time in the right atrium and ventricle proximal to the obstruction

8 *Surgical* lysis of the stenosis is of considerable therapeutic value and is fairly safe in *experienced* hands

B **Pulmonary Infundibular Stenosis** The findings are essentially the same as described for valvular stenosis except

1 Poststenotic dilatation is absent

2 The systolic murmur and thrill are lower

3 Right ventricular left parasternal lift tends to be absent especially if the stenosis is low

4 Cardiac catheterization may reveal three different zones of pressure and it may be necessary to withdraw the catheter considerably for the tip to find itself in the right ventricle proper

C **Pulmonary Stenosis with Ventricular Septal Defect** The findings are those of pulmonary stenosis with a left-to-right shunt through the ventricular septal defect The diagnosis is especially well established by cardiac catheterization with the usual expected findings of pressure and O concentration

D **Pulmonary Stenosis with Atrial Septal Defect** The picture is one of both defects If the pulmonary stenosis is severe and the right atrial pressure is high there will be a right to left shunt with the pat[illegible]

[illegible] foramen ovale which readily allows a flow of blood from right to left Cyanosis usually occurs late and progresses with age and with exertion The diagnosis is usually confused with tetralogy of Fallot Since the Blalock operation is not to be performed in pulmonary sten[illegible] *Angiocardiography* w[illegible] ere is a right to left sh[illegible]

Tetralogy of Fallot

1 Tetralogy of Fallot is one of the most common syndromes of the multiple congenital defects It is found [illegible]

a Ventricular septal patency

b Pulmonary stenosis

c Anatomic or functional overriding of the aorta

d Right ventricular hypertrophy

The [illegible]

2

It t[illegible] progress for several years

before reaching a relatively fixed state. Cyanosis is due to shunting of venous blood low in O_2 content from the right ventricle directly into the aorta, with distribution throughout the systemic circulation (Fig 160). *Secondary polycythemia* is a usual manifestation.

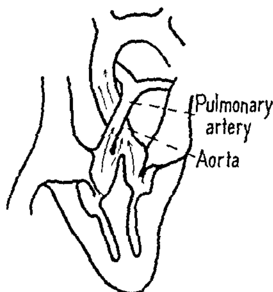


FIG 160 —Diagram showing how *overriding of the aorta in tetralogy of Fallot* produces cyanosis unoxygenated blood enters the aorta from the right ventricle in addition to oxygenated blood from the left ventricle

3 *Signs*

a The jugular pulsations are not necessarily altered. The *a* wave is increased only slightly if at all.

b A *systolic murmur* and *thrill* are elicited best in the second, third or fourth intercostal space just to the left of the sternum. A thrill is not always present, but a murmur can always be heard.

c The *pulmonic second sound* is practically always single and clear and is usually loud. It represents the first or aortic component without a pulmonic second component. It tends to be loud because the aorta is shifted more anteriorly when it is displaced.

d Precordial palpation reveals *right ventricular enlargement* with lifting of the precordium with the heart beat.

e *Precordial bulging* due to right ventricular enlargement is usually noted.

f *Impairment of mental and physical growth* is common, the degree being related to the severity of the defect.

g *Squatting* is observed frequently.

h Manifestations of *congestive heart failure* are commonly observed.

4 *Roentgenographic studies* reveal enlargement of the right ventricle

tendency for the apex of the heart to appear displaced upward and to the left the left edge of the cardiac shadow to be concave in many instances due to absence of the pulmonary conus and artery, small or absent aortic knob and avascularity or clarity of the pulmonary fields (Fig 161) ... and its branches are small or absent ... may be noted especi- (Fig 162)

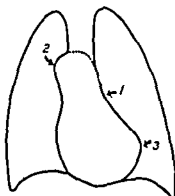


Fig 161 —The anteroposterior silhouette in tetralogy of Fallot Three important characteristics are shown (1) The extreme concavity of the left margin of the heart shadow at the region of the pulmonary conus This is due to atresia of the pulmonary artery and conus and dextroposition of the aorta (2) The aortic knob and aorta are on the right (3) *The right ventricular enlargement literally lifts the heart up and displaces* the apex and interventricular knob or groove cephalad This lifting effect is due to the fact that the right ventricle rests on the diaphragm When it enlarges it displaces the heart cephalad

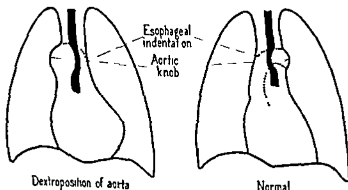


Fig 162 —

5 The *electrocardiogram* shows extreme right axis deviation and large R waves in V_1 and large S waves in V_5 and V_6 which are indicative of right ventricular hypertrophy. The P waves are usually high, wide and peaked, a common finding in congenital heart disease with right ventricular hypertrophy.

6 *Cardiac catheterization* reveals an increase in pressure in the right ventricle and an increase in O_2 content in the right ventricle. The catheter may be passed directly into the left ventricle or into the aorta, thus demonstrating the ventricular defect and anterior displacement of the aorta. The lower pressure in the pulmonary artery than in the right ventricle is often demonstrable, as indicated previously for pulmonary stenosis. Because of atresia of the pulmonary artery, the vessel may be too small for the catheter to enter for pressure recordings.

7 *Angiocardiography* reveals rapid filling of the aorta directly from the right ventricle, with relatively little circulation of the radiopaque medium into the pulmonary vessels.

8 *Surgical therapy* consists in shunting blood from a systemic artery in the chest into a pulmonary artery distal to the point of stenosis. This improves aeration of blood and thus ameliorates the state of cyanosis. The operation results in a shunting procedure similar to that for patent ductus arteriosus. Although the patient is improved immediately, his life may be prolonged for only a few years. Follow up studies are required to determine permanent postoperative effects on life expectancy and cardiac function. It is well to remember that such a patient will continue to have serious heart disease.

Eisenmenger's Complex

1 This syndrome of multiple congenital cardiac anomalies is not common. It represents about one per cent of the cardiac congenital defects. The diagnosis is frequently made but is rarely substantiated. It is frequently referred to as the *tetralogy of Eisenmenger* because it consists of

- *a* Ventricular septal defect
- *b* Anatomic or functional overriding of the aorta
- *c* Pulmonary arterial dilatation
- *d* Right ventricular hypertrophy

The clinical picture is described by the following characteristics

2 Moderate *cyanosis* occurs fairly late but is not as severe or as progressive as *in tetralogy of Fallot*. *It is central in origin*. There may be mild to moderate but never severe clubbing of the fingers and toes.

3 *Signs*

- *a* The cardiac impulse is *tapping* and the conus area and precordium manifest *lifting* with the heart beat due to enlargement of the conus area and right ventricle.
- *b* There may be *palpable pulsations of the pulmonary artery*.
- *c* A systolic *murmur* and *thrill* are found in the third intercostal space near the left margin of the sternum.

d *The pulmonary second sound* is split as in the normal subject, but the second or pulmonary valvular component is accentuated

4 *Roentgenographic studies* reveal right ventricular enlargement, a prominent pulmonary conus and dilatation of the pulmonary arteries with fairly well defined peripheral vascular shadows The pulmonary vascular pulsations are essentially normal

5 The *electrocardiogram* manifests right ventricular hypertrophy

6 *Cardiac catheterization* shows an increase in pressure in the right ventricle and in the pulmonary arteries The blood shows a decrease in oxygen content in the right ventricle pulmonary arteries, right atrium and venae cavae The blood in the pulmonary veins and left atrium are of normal O_2 saturation Blood in the aorta is less saturated with O_2 than normal blood due to the fact that blood is pumped into the aorta from the right ventricle In some instances the catheter may be shown to enter the aorta and the left ventricle an observation which is of *considerable value in* definitely establishing the diagnosis

7 *Angiocardiography* shows the radiopaque material to enter the aorta and pulmonary artery simultaneously without entering the left atrium before filling of the aorta

8 *Differential diagnosis* becomes especially important in this syndrome because the Eisenmenger complex is so frequently erroneously diagnosed Among the clinical states falsely considered the Eisenmenger *complex are*

a *Idiopathic* pulmonary hypertension with reversal flow through a patent foramen ovale atrial *septal defect or* ventricular septal defect This is the most common cause for error

b *Pulmonary* stenosis with poststenotic dilatation and atrial septal defect

9 *Surgical intervention* is not indicated in this congenital defect

Tricuspid Atresia

1 Tricuspid atresia is *relatively rare* representing about three per cent of congenital cardiac defects It has *essentially* the following significant diagnostic manifestations

2 *Cyanosis* of central origin

3 *Diminished pulmonary blood flow* manifested roentgenographically by absence or *reduced* pulmonary vascular shadows and pulsations

4 *Roentgenographic evidence* of left ventricular hypertrophy and absence of a small right ventricle and pulmonary conus and arteries

5 *Electrocardiographic evidence* of left ventricular hypertrophy This is the only cyanotic type of *congenital cardiac* defect in which there is such an electrocardiographic *manifestation* This is due to the rudimentary right ventricle

6 *Cardiac catheterization* shows an atrial septal defect with right to left shunting The catheter may enter the left atrium

7 *Angiocardiography* reveals immediate filling of the left atrium and ventricle with little or no demonstrable filling of the right ventricle and

5 The *electrocardiogram* shows extreme right axis deviation and large R waves in V_1 and large S waves in V_5 and V_6, which are indicative of right ventricular hypertrophy. The P waves are usually high, wide, and peaked, a common finding in congenital heart disease with right ventricular hypertrophy.

6 *Cardiac catheterization* reveals an increase in pressure in the right ventricle and an increase in O_2 content in the right ventricle The catheter may be passed directly into the left ventricle or into the aorta, thus demonstrating the ventricular defect and anterior displacement of the aorta The lower pressure in the pulmonary artery than in the right ventricle is often demonstrable, as indicated previously for pulmonary stenosis Because of atresia of the pulmonary artery, the vessel may be too small for the catheter to enter for pressure recordings

7 *Angiocardiography* reveals rapid filling of the aorta directly from the right ventricle, with relatively little circulation of the radiopaque medium into the pulmonary vessels

8 *Surgical therapy* consists in shunting blood from a systemic artery in the chest into a pulmonary artery distal to the point of stenosis This *improves aeration of blood and thus ameliorates the state of cyanosis* The operation results in a shunting procedure similar to that for patent ductus arteriosus Although the patient is improved immediately, his life may be prolonged for only a few years Follow-up studies are required to determine *permanent postoperative effects on life expectancy and cardiac function* It is well to remember that such a patient will continue to have serious heart disease

Eisenmenger's Complex

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b Anatomic or functional overriding of the aorta
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d Right ventricular hypertrophy

The clinical picture is described by the following characteristics

2 Moderate *cyanosis* occurs fairly late but is not as severe or as progressive as in tetralogy of Fallot It is central in origin There may be mild to moderate, but never severe clubbing of the fingers and toes

3 *Signs*

a The cardiac impulse is *tapping*, and the conus area and precordium manifest *lifting* with the heart beat due to enlargement of the conus area and right ventricle
b There may be *palpable pulsations of the pulmonary artery*
c A systolic *murmur* and *thrill* are found in the third intercostal space near the left margin of the sternum

through the lungs and left atrium The heart is quickly emptied of the contrast material with little or no filling of the left cardiac chambers Curiously enough, the pulmonary vessels are not well visualized by angiocardiography

7 The *most successful operation*, if the patient survives it, consists in anastomosis of the right superior pulmonary vein into the right atrium creation of an atrial septal defect and anastomosis of the end of a subclavian artery to the distal end of a pulmonary artery

Coarctation of the Aorta

These *defects* constitute about 10 per cent of all congenital anomalies ...

...

2 The *adult type* which is a narrow area of constriction usually just ... type is much more common than ...

1 *Hypertension* systolic and diastolic in the vessels having their origin proximal to the site of narrowing (Fig 163) The systolic blood pressure in the arteries originating below the site of constriction is low whereas the *diastolic* blood pressure is usually elevated but to a lesser degree than in the vessels originating proximal to the site of constriction To detect these blood pressure changes which are extremely important in diagnosis one must determine the arterial pressure in both the upper and lower extremities

2 ...

... to pulsate more vigorously than normal and to be more easily palpated Smooth erosion of bone is noted *roentgenographically* particularly near the vertebral *ends of the* ribs where the intercostal arteries course under the ribs to run along their inferior and internal margins This produces a scalloping of the ribs

4 The *aortic knob* is reduced or absent as noted by roentgenographic study

5 *Left ventricular enlargement* is almost invariabl...

... is to be incompatible with life and it is usually *too* severe to permit long life *The adult t*...

pulmonary arteries The latter disturbance varies in degree with the extent of the tricuspid atresia The bronchial arteries which are mainly responsible for the pulmonary circulation may be demonstrated by angiocardiography

8 The same type of *surgical shunting* operation is indicated for this type of defect as for tetralogy of Fallot

Transposition of the Great Vessels

1 This defect constitutes 8 to 10 per cent of all the congenital abnormalities of the heart Because it is relatively common its clinical characteristics should be known

2 *Dyspnea* is always present but squatting seems to offer little relief *Coughing* may be pronounced and distressing

3 *Signs*

- a *Cyanosis* is always present from birth It is aggravated by crying coughing feeding and exposure to cold The lower part of the body may be less cyanosed than the upper part *Secondary polycythemia* seen in all cyanotic types of congenital defects is observed Clubbing of the fingers and toes is present and may be especially severe
- b *Congestive heart failure* is common
- c A *systolic murmur and thrill* are found in the third interspace near the left margin of the sternum
- d *Palpation of the precordium* reveals lifting over the right ventricular and conus areas and pulmonary arterial pulsations due to enlargement of these structures

4 *Roentgenographic studies* are especially important revealing *extreme pulmonary plethora* and moderate pulmonary vascular pulsations *in a cyanotic child* are almost diagnostic in themselves The aortic and pulmonary arterial shadows are narrow on the anteroposterior view and become especially wide in the oblique and lateral views The presence of a *concave left middle segment of the anteroposterior view with pulmonary plethora* is especially important Occasionally there is a *long* bulge or convexity to the left middle segment in the anteroposterior view in these patients

5 *Cardiac catheterization* reveals an elevation in pressure within the right ventricle and pulmonary arteries The O_2 saturation of the blood is *reduced throughout the cavities studied* Passage of the tip of the catheter into the aorta and at times also into the pulmonary artery is diagnostic of the defect If there is a ventricular septal defect and a left to right shunt the blood in the pulmonary artery will contain more O_2 than that in the aorta and peripheral arteries There may also be an atrial septal defect Although shunting through the ventricular septal defect may be right to left or left to right that through the atrial septal defect is right-to-left

6 *Angiocardiography* reveals immediate filling of the aorta from the right ventricle without previous circulation of the radiopaque material

through the lungs and left atrium The heart is quickly emptied of the contrast material with little or no filling of the left cardiac chambers Curiously enough the pulmonary vessels are *not* well visualized by angiocardiography

7 The *most successful operation*, if the patient survives it, consists in anastomosis of the right superior pulmonary vein into the right atrium creation of an atrial septal defect and anastomosis of the end of a subclavian artery to the distal end of a pulmonary artery

Coarctation of the Aorta

These defects constitute about 10 per cent of all congenital anomalies There are two types

1 The *infantile type* which consists in narrowing of the whole isthmus or that portion between the left subclavian artery and the ductus arteriosus It should be remembered that in fetal life the isthmus carries very little blood

2 The *adult type* which is a narrow area of constriction usually just proximal to the ductus arteriosus This type is much more common than the [illegible]

[illegible] vessels having their origin proximal to the site of narrowing (Fig 163) The systolic blood pressure *in the arteries originating below the site of constriction is low* whereas the diastolic blood pressure is usually elevated but to a lesser degree than in the vessels originating proximal to the site of constriction To detect these blood pressure changes which are extremely important in diagnosis one must determine the arterial pressure in both the upper and lower extremities

2 [illegible]

3 [illegible]

scapt [illegible]

to pi [illegible]

Smoo[illegible] rosion of bone is noted *roentgenographically* particularly near the vertebral ends of the ribs where the intercostal arteries course under the ribs to run along their inferior and internal margins This produces a scalloping *of the ribs*

4 The *aortic knob* is reduced or absent as noted by roentgenographic study

5 *Left ventricular enlargement* is almost invariably present

6 *Angiocardiography* makes localization of the [illegible]

[illegible] is incompatible with life and it is usually too severe to permit long life The *adult type* may be so slight as not to alter the cardiac state to any demonstrable extent Left ventricular congestive heart failure is a common complication which must be *treated* in the usual manner

The *management* of coarctation of the aorta is essentially the same as that for any congenital cardiac defect It should be governed by the severity of the defect Limitation of physical activity, good *hygienic measures*

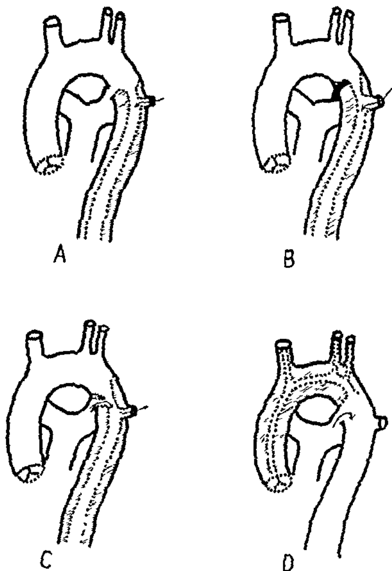


Fig 163 — Illustration of various types of coarctation of the aorta Part A shows atresia of the aorta distal to the ductus arteriosus (duct of Botalli) The duct is patent a fairly common association because the high pressure in the proximal portion of the aorta tends to prevent the ductus from closing The arrows show how blood enters the narrowed aorta via the vertebral arteries or other branches Part *B* shows the same type of coarctation with the ductus arteriosus closed Part *C* shows an atresia of the aorta as in Part *A*, except that it begins proximal to the ductus arteriosus The ductus ... Part D shows atresia or coarctation of the proximal portion of the

and adequate cardiac observation from time to time are certainly indicated. The physician should prevent the development of a "cardiac invalid" or a "cardiac neurosis." Surgical management is still in the stage of development, being adequately performed by few surgeons.

In general it is important to indicate that the foregoing discussions of the more common congenital cardiac defects have necessarily been brief. All of the defects have not been discussed and some of the variations have been omitted. The medical literature must be studied to learn more of the details of the methods employed for the study of congenital heart disease, for the disturbances in oxygenation of blood and in hemodynamics, and for surgical management.

OTHER ETIOLOGIC TYPES OF HEART DISEASE

Other etiologic types of heart disease include: (1) pulmonary, (2) traumatic, (3) neoplastic, (4) nephritic, (5) toxic, etc. Discussion of these may be found in other monographs on cardiology and in the general medical literature.

CONGESTIVE HEART FAILURE

Because of its frequency and occurrence as a complication in any type of heart disease, congestive heart failure is briefly presented here.

It is assumed that all students have acquainted themselves with the ph...

... basis of congestive failure is by no means settled or simple. There ...

The ... here

the ... he severity of

fail... severe acute

mer... The treat...

... following paragraphs is for Functional Class IV congestive heart failure. For less severe episodes the treatment is varied accordingly.

The therapy is essentially as follows:

1. **Rest**.—Without a doubt the most important therapeutic agent at the physician's command is rest, both mental and physical. The rest should be *absolute* and not include bathroom privileges. A few minutes of exercise in order to walk to the bathroom can overcome the benefits accomplished by many hours of bed rest. The ... the complete removal of domestic ... bilities. The physician should de...

attendants to effect these aims A good nurse prompt and constant attention a radio *pleasant* and *nonexciting visitors* in limited numbers and for a short time and peaceful surroundings are of extreme value It is only by meticulous attention to details that good results are obtained After all, a patient with severe congestive heart failure represents a medical emergency and is bordering on death therefore it is not advisable to be superficial Attention to minute details in therapy is imperative

2 **Environment** —The patient s environment or atmosphere of the room should be made comfortably cool with the use of air-conditioning if possible The oxygen tent will serve in this capacity, as well as in the supply of oxygen if air-conditioning is not available This is particularly important during heat waves and in subtropical and tropical environments It is important not to allow the room air to become warm and humid, since this makes thermal regulation difficult and increases the cardiac output and work The increase in work is equivalent to that associated with exercise which is known to be detrimental The rate of heat production is already elevated by the work associated with the dyspnea and apprehension

3 **Morphine** —This is one of the most important drugs in medicine and especially in congestive heart failure Adequate doses must be used however at least ¼ grain (15 mg) subcutaneously as soon as the patient is placed in bed This removes the anxiety panic and fear of death which accompanies these episodes especially the first one The injections should be repeated at frequent intervals if necessary of course avoiding depression of respiration

4 **Oxygen** —This is best administered in an oxygen chamber but may be done *fairly well* by a mask tent or nasal catheter The method least annoying should be employed The method should be changed from time to time for the patient s comfort The inspired air should contain about 55 per cent oxygen if possible This should be checked quantitatively if possible

5 **Phenobarbital** should be used *only* when necessary to reduce anxiety and to produce sleep but not empirically The dose given should vary with the patient grain 1½ (90 mg) tablets once twice or three times daily, that is enough to produce the desired pharmacologic effects

6 **Sodium intake** should be restricted to a minimum not over 1 7 grams daily To accomplish low sodium levels sodium-containing drugs should be avoided

7 **Diet** —It is advisable to administer no food for the first twenty four to thirty six hours The food allowed should be soft low in residue free from added salt and intrinsically low in salt Cereals milk soft boiled eggs vegetable purees etc may be used The baby foods low in salt may be used Frequent small feedings should be employed never allow the patient to consume a large meal Foods known to produce gastro-intestinal disturbances should be avoided The Karrel diet only 800 cc of milk per day, may be valuable

8 **Fluids** —Water should be allowed *ad libitum* Patients who are not eating very much salt will not drink very much anyway Avoid cold drinks, as there is a possibility of reflex coronary vasoconstriction from the

local cooling Tap water at room temperature is best There is no apparent adequate reason for forcing water if sodium is restricted

6 **Digitalis** is an important drug It should be administered in full doses and rapidly

about 1 U S P unit

italize fully the

immediately at the end of eight hours one-half of the remaining dose is given and then 2 units every eight hours until full digitalization is reached *The patient should be examined before each dose is given in order to avoid overdigitalization and to obtain the desired effects* For example, if a patient weighs 160 pounds he will require about 20 units of digitalis to digitalize

given patient will require Administer the *correct amount* as determined by the clinical results One unit daily or more will be necessary for maintenance of digitalization The digitalis leaves or the pure glucosides, such as digitoxin digoxin or lanatoside C may be employed whole-leaf orally is preferable Subcutaneous or intravenous administration is employed in the severe and very acute failure The dosage may be smaller If the patient is checked before each dose, no serious error can occur The importance

in

ployed Should diuresis fail to occur the drug should be stopped Examine the urine at frequent intervals to avoid renal damage from the mercury One to 2 cc intravenously during the period of acute failure may be necessary even before full digitalization has been effected This is a good drug It is sometimes possible to maintain the patient free of edema by daily subcutaneous administration of 0.25 to 0.50 cc of a mercurial diuretic Patients can be taught to administer this to themselves at home just as diabetic patients are taught to use insulin

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of are large and are producing discomfort Removal of the fluids should be done slowly Their removal is often followed by diuresis apparently from improvement of the circulation

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3. **Morphine.**—This is one of the most important drugs in medicine and especially in congestive heart failure. Adequate doses must be used, however, *at least ¼ grain (15 mg.) subcutaneously as soon as the patient is placed in bed.* This removes the anxiety, panic and fear of death which accompanies these episodes, especially the first one. The injections should be repeated at frequent intervals if necessary, of course avoiding depression of respiration.

4. **Oxygen.**—This is best administered in an oxygen chamber, but may be done *fairly well* by a mask, tent or nasal catheter. The method least annoying should be employed. *The method should be changed from time to time for the patient's comfort. The inspired air should contain about 55 per cent oxygen, if possible. This should be checked quantitatively, if possible.*

5. **Phenobarbital** should be used *only* when necessary to reduce anxiety and to produce sleep, but not empirically. The dose given should vary with the patient; grain 1½ (90 mg.) tablets once, twice or three times daily, that is, enough to produce the desired pharmacologic effects.

6. **Sodium intake** should be restricted to a minimum, not over 1.7 grams daily. To accomplish low sodium levels, sodium-containing drugs should be avoided.

7. **Diet.**—It is advisable to administer no food for the first twenty-four to thirty-six hours. The food allowed should be soft, low in residue, free from added salt and intrinsically low in salt. *Cereals, milk, soft boiled eggs, vegetable purees, etc. may be used. The baby foods low in salt may be used.* Frequent small feedings should be employed; never allow the patient to consume a large meal. Foods known to produce gastro-intestinal disturbances should be avoided. The Karrel diet, only 800 cc. of milk per day, may be valuable.

8. **Fluids.**—Water should be allowed *ad libitum*. Patients who are not eating very much salt will not drink very much anyway. Avoid cold drinks, as there is a possibility of reflex coronary vasoconstriction from the

Sedatives and *laxatives* should be used only as indicated but they are not to be employed routinely. This is also true for any other drugs that may be necessary.

Diet.—The patient's diet should be limited to foods which do not produce dyspepsia. It should be well balanced, not necessarily limited to any special foods, but restricted only in sodium if the patient has a tendency to develop congestive heart failure. It should be bland; feeding should be frequent, and the patient should be advised to eat especially fresh fruits and vegetables. He should reduce his weight if he is obese. Patients frequently have a tendency to gain weight when they are placed at rest and this of course should be avoided. Such patients should never eat heavily and certainly should never exercise after meals. Frequent small feedings and eating slowly are to be advocated.

Tobacco, Caffeine Beverages and Alcohol.—The rules for tobacco, caffeine beverages and alcohol are outlined in the discussion on the treatment of coronary occlusion. The patient should be advised to refrain completely from the use of tobacco, to restrict himself to one caffeine beverage daily, and to use alcohol sparingly. Alcohol should not be prescribed to the patient except for its tonic effect, since there is no concrete evidence that it produces coronary dilatation.

General Hygienic Measures.—The usual general hygienic measures should be enforced. The patient should be advised, once he starts to ambulate, not to overexert himself and not to become disturbed mentally or physically. He should obtain his full number of hours of sleep and have a period of rest after his noon meal with the possibility of a short rest during the middle of the morning and the middle of the afternoon if his state of angina pectoris warrants this. He should not rush during the course of the day nor work excessively. He should remain free from tension and should work in a relaxed state. H[illegible]
long annual vacation
nature should be reso
producing unhappiness
have the patient relaxed and happy. If he has any systemic diseases they should be properly treated. Infections such as those of the gums or tonsils should be properly handled. The patient should see his physician at regular intervals to make sure that his therapeutic regimen is being followed and that any complications such as congestive heart failure which may develop are treated early.

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as well. T
definite [illegible] patient and which may have a detrimental side effect is to be avoided. The patient should not be overtreated with drugs since there are no known specific drugs of value for the cure of this disease. Physicians frequently make the mistake of overtreating the patient, administering numerous drugs and pills during the course of a day; these in themselves produce unhappiness and disturbances in the

TREATMENT OF ANGINA PECTORIS

Angina pectoris is another common cardiac problem which the cardiologist, internist, and general practicing physician encounter in cardiology. The therapeutic procedures outlined briefly in the following paragraphs are concerned with the average patient and are not intended to be a detailed and complete review of the problem.

Rest.—Once the diagnosis of angina pectoris has been made, it is probably best for the patient to be placed in bed for a period of four weeks. Apparently more rapid and definite benefits in therapy may be achieved by this procedure, since it removes the patient from his daily routine and thus permits him to eliminate all problems of a psychologic as well as physical nature which he encounters in his normal daily activities. Most patients with angina pectoris have various kinds of mental and physical stresses. With bed rest these cease and the patient has an opportunity for reflection and realization that a modification of his life is necessary if he is to benefit from therapy and live in fairly good health for several more years. During this period of bed rest it is possible for the physician to evaluate the general medical state of the patient by careful medical inventory, to try various forms of therapy, and to work with the patient in planning future therapy.

Drugs.—Few drugs are effective in the management of angina pectoris. Nitroglycerin, $\frac{1}{200}$ gr. or more administered sublingually, is probably the *drug of greatest value*. The patient may use it to avoid pains as frequently as desired without acquiring tolerance to the drug. He should be advised to carry nitroglycerin with him at all times and to take it as soon as the pain occurs rather than delaying until the pain has reached maximal intensity.

Vasodilators.—As mentioned in the preceding discussion of the management of coronary occlusion, there are no reliable vasodilators. Aminophylline may be tried in doses of 0.1–0.2 Gm. (1.5–3 gr.) three or four times daily, administered for two or three successive days and then discontinued for one or two days in order to avoid tolerance. Should gastrointestinal disturbances appear, the drug should be discontinued. Other *vasodilators have been advocated, such as papaverine, peritrate,* and khellin, but results have generally been poor and have varied considerably from patient to patient. If beneficial, their administration should be continued, but if they produce unfavorable side effects, they should, of course, be discontinued promptly.

Sedatives.—Sedatives should be employed sparingly. Barbituric acid derivatives such as *phenobarbital at bedtime is advocated only for* patients with insomnia and for patients who are restless and irritable, and is not recommended as a routine procedure. Sodium-containing drugs should be avoided if the patient has congestive heart failure or a tendency toward it. If the patient is free from failure, it is possible to use sodium-containing drugs when needed. Patients should not be oversedated, as is unfortunately done by some physicians.

Vitamins and *laxatives* should be used only as indicated but they are not to be employed routinely. This is also true for any other drugs that may be necessary.

Diet.—The patient's diet should be limited to foods which do not produce dyspepsia. It should be well balanced, not necessarily limited to any special foods but restricted only in sodium if the patient has a tendency to develop congestive heart failure. It should be bland; feeding should be frequent and the patient should be advised to eat especially fresh fruits and vegetables. He should reduce his weight if he is obese. Patients frequently have a tendency to gain weight when they are placed at rest and this of course should be avoided. Such patients should never eat heavily and certainly should never exercise after meals. Frequent small feedings and eating slowly are to be advocated.

Tobacco, Caffeine Beverages and Alcohol.—The rules for tobacco, caffeine beverages and alcohol are outlined in the discussion on the treatment of coronary occlusion. The patient should be advised to refrain completely from the use of tobacco, to restrict himself to one caffeine beverage daily and to use alcohol sparingly. Alcohol should not be pre-

He should obtain his full number of hours of sleep and have a period of rest after his noon meal with the possibility of a short rest during the middle of the morning and the middle of the aftern

He should take frequent short vacations and one long annual vacation. Any mental nature should be resolved they are producing unhappiness to t made to have the patient relaxed and happy. If he has any systemic diseases they should be properly treated. Infections such as those of the gums or tonsils should be properly handled. The patient should see his physician at regular intervals to make sure that his therapeutic regimen is being followed and that any complications such as

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mental side The patient should not be overtreated with drugs since there are no known specific drugs of value for the cure of this disease. Physicians frequently make the mistake of overtreating the patient, administering numerous drugs and pills during the course of a day; these in themselves produce unhappiness and disturbances in the

patient The physician should make every effort to encourage the patient and see that he does not become a so-called 'cardiac neurotic' since these patients, as a rule, are anxious and frequently suffer from an anxiety state anyway

Exertion —The patient should be advised not to exert himself to any extent that is known to produce anginal pain He should be instructed that any procedure or any action on his part which he knows will produce pain should be avoided He should be made to walk as little as possible if walking produces pain If heavy meals precipitate pain he should be instructed to avoid them Patients are also instructed not to test themselves by the use of exercise to learn whether or not they are improving They should avoid walking up steep grades or exerting themselves particularly at high altitudes They may be allowed airline travel provided there is no oxygen deficiency brought about by extreme high flying in nonpressurized cabins The occupation should be modified if necessary, to obtain sufficient relaxation and to avoid annoyances which tend to produce anginal pain Many of the details in therapy are solved by the patient and the physician as they follow the disease Each patient must be individualized

TREATMENT OF CORONARY OCCLUSION

The management of coronary occlusion is one of the principal problems confronting the cardiologist and the internist It compares in importance with congestive heart failure and angina pectoris these three constituting the major problems in therapy associated with cardiovascular diseases *It is intended in the discussion to follow to present only briefly some of the* more pertinent principles employed in the treatment of patients with coronary occlusion

Mental and Physical Rest —As in the management of congestive heart failure proper rest both mental and physical is essential in the management of a patient with coronary occlusion Every effort should be made

the patient is comfortable and free from
nurses physician and others attending
ing his environment pleasant Visitors

should be limited to one or two who can bring comfort and relaxation to the patient He should divorce himself immediately from all business and other worries All of this can be achieved at the patient's home particularly if his family is not large and the facilities for proper care are available and especially if he cannot readily afford hospital costs When necessary the patient should be moved to a private room in a hospital

Environment of the Patient's Room —As in congestive heart failure the environmental atmosphere of the patient's room should be made comfortable and should certainly be free from overheating or high humidity This may be achieved by means of air-conditioning proper ventilation of the room or, when this is not possible by means of the oxygen tent The cool atmosphere of an oxygen tent particularly in tropical and subtropical

environments often offers more comfort and benefit to the patient than the oxygen itself Every effort should be made to avoid overheating of the room by crowding of people or by tendency to prevent adequate ventilation

Morphine —One of the most valuable drugs available to the physician in the management of a patient with coronary occlusion is morphine The physician should use adequate doses immediately at least a quarter of a grain subcutaneously In the event that the pain is extremely severe morphine may be given intravenously If the pain is not alleviated within an adec The main difficulty hich may be of some ave respiratory disturbances due to senility It is important not to use small doses since the pain of coronary occlusion is one of the most severe that man experiences

Oxygen The use of oxygen should be routine if the patient shows evidences of impairment of respiration congestive heart failure or cyanosis It is desirable to administer about 50 per cent oxygen preferably in an oxygen room or if that is not available by means of an oxygen tent both because of the oxygen and the air-conditioning influences from the tent If that is not available then nasal oxygen or oxygen mask may be used Administration should be continued as long as is necessary

Sedation.—Care should be taken not to oversedate the patient Many physicians today tend to prescribe too much of barbituric acid derivatives and other sedatives and to keep the patient in an oversedated state Sedatives should be used only to produce relaxation and to overcome insomnia If the patie sedatives are not nece effects Sodium-cont for sodium to predispo to edema formation

Vasodilators —There are no well-establ dilators Aminophylline

distress particularly if there is It should be administered slowly the patient being carefully observed as the drug is being injected into the vein One-tenth to three tenths grain (1 3 gr) three or four times daily may be administered orally to improve the coronary circulation recession skipping Acquired tolerance the drug produces Some physicians er this to be more be tried but it is as a rule, not especially promising In general there are no reliable coronary vasodilators available

Diet —During the early stages of infarction the patient should receive little to eat If he desires food he may be limited to liquids followed in

two or three days by a soft bland diet and then may be allowed to return gradually to a regular diet Sodium in the diet should be avoided because of its tendency to predispose to edema formation Water may be allowed as desired by the patient

Digitalis —Digitalis is indicated only for congestive heart failure auricular fibrillation auricular tachycardia or auricular flutter It is not to be used unless definitely indicated If the patient has mild congestive heart failure other measures should be tried before digitalis This is important since digitalis predisposes to the development of cardiac irregularities of ventricular origin which may result in ventricular tachycardia and possibly ventricular fibrillation However when the patient definitely has congestive heart failure and it is progressing digitalis must be used in the usual manner as described previously for the management of congestive heart failure

Quinidine —Quinidine is not to be used routinely in all patients with coronary occlusion It should be employed however if the patient presents *cardiac irregularities such as paroxysmal tachycardia frequent* premature contractions auricular flutter auricular fibrillation and certainly irregularities of ventricular origin An occasional premature beat of ventricular origin does not necessarily justify resorting to quinidine *however if they persist or show a tendency to become multifocal multiple* or frequent quinidine should be administered promptly The dose should be 0 2 0 4 Gm (3 6 gr) every two to three hours or it may be increased depending upon the type of irregularity and the clinical state It should be continued until the mechanistic disturbance is corrected then the dose should be reduced and a maintenance dose of 0 2 Gm every three or four hours for two or three weeks should be given Should the patient present some of the toxic side effects of quinidine the drug should be discontinued immediately As a rule this is not necessary

Anticoagulants Heparin and dicumarol as well as some of the newer anticoagulants have been advocated by some to be used routinely in coronary occlusion This appears to be unnecessary and certainly not justified by the data obtained to date They should be reserved only for patients who have definite thrombophlebitic disturbances Even when thrombophlebitic phenomena are established these anticoagulants have questionable value so that the physician must decide whether veins are to be ligated if the vein presenting the phlebothrombosis can be identified or if anticoagulants are *to be employed* At present *the management* of thrombophlebitic phenomena is unsatisfactory despite published reports to the contrary Whenever anticoagulants are employed it is important that they be used cautiously and supported by adequate determinations of prothrombin time

Other Drugs —Should the patient develop cardiac distress with profuse sweating and evidences of peripheral circulatory collapse the tendency is to use many of the so-called stimulants Coramine metrazol caffeine sodium benzoate and others have been employed Most of them are probably more deleterious than useful Patients are frequently made worse

by the therapy, so that it becomes difficult to evaluate the results of the drugs or distinguish their effects from the state of the disease itself. It is important therefore not to complicate the clinical picture by using such drugs particularly if they are not likely to be beneficial.

Shock.—Patients with acute coronary occlusion are prone to develop circulatory collapse and so-called medical shock. There is no dependable method available today for the management of this state. Infusions of plasma and transfusions have been tried but results have not been particularly encouraging. At the present time the most effective procedure appears to be a slow infusion of *nor-epinephrine* to maintain the blood pressure not at exceedingly high levels but at levels sufficiently high to insure adequate perfusion of the tissues by blood and for adequate glomerular filtration. Patients who had high blood pressure prior to the occlusion should have their blood pressure elevated by nor-epinephrine to higher levels than patients whose blood pressure was normal prior to the occlusion. The infusion [illegible]

regulated b[illegible]

discontinue[illegible]

the blood pressure decline when the infusion is discontinued it should be reinstituted immediately. As a rule when such infusions are employed for many hours they should be discontinued gradually. Sudden cessation of infusions of nor-epinephrine often results in a rapid return to the shock levels of blood pressure whereas a gradual reduction in the rate of infusion permits the regulating blood pressure mechanisms of the body to assume gradually the function of maintaining blood pressure. Failure with the use of nor-epinephrine in maintaining blood pressure in the shock-like state associated with coronary occlusion has usually been due to [illegible] use of [illegible]

us[illegible]

th[illegible]

co[illegible] degree of bed rest is varied but six weeks appears to be an advisable period. Patients who find it difficult to use a bedpan may be permitted to use a commode placed next to the bed. The patient should be assisted in and out of bed for use of the commode by attendants. Some people experience less distress [illegible]

he is not actually

Tobacco and Caffeine Beverages.—Tobacco should be prohibited in a
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mine which patient may be injured by tobacco it is advisable to eliminate the use of tobacco completely in all patients. Caffeine beverages should be permitted only in small quantities because they tend to produce restlessness, insomnia and excitement in the patient and not because of any direct action of the caffeine beverages on the heart. They are reduced to approximately one caffeine beverage per day. Alcohol has been prescribed because of the impression that it has coronary vasodilative influences. There is no evidence to support such benefits from alcohol. Probably its only value is its tonic effect, that is, for its psychologic influences rather than any direct pharmacologic action. Certainly the patient may be allowed to have an occasional alcoholic beverage but it should not be prescribed for those who do not desire it.

Laxatives.—Mild laxatives should be given particularly to avoid straining at stool. Patients who have had large quantities of morphine become constipated and therefore the use of mild laxatives is indicated. Laxatives that tend to produce gastrointestinal colic or any discomfort to the patient should obviously be avoided.

Nursing.—Good nursing care, not necessarily by professional nurses but by members of the family, practical nurses or friends, is essential in the management of such patients, particularly in the acute stages of illness. Making the patient comfortable, maintaining morale and preventing the patient from becoming discouraged and excessively frightened by his cardiac disease are all extremely important.

Course of Convalescent Management.—The value of periodic follow-up examinations in order to avoid future infarcts and to detect early manifestations of angina pectoris or congestive heart failure cannot be overemphasized. During this follow-up period the physician should avoid making a cardiac neurotic of his patient but should keep his patient properly advised and encouraged to carry on as normal a life as his cardiac reserve will permit. Should complications or sequelæ develop, such as congestive heart failure or angina pectoris, careful follow-up studies will permit early recognition and adequate management before these states become irreversible and difficult to manage.

CHAPTER 5

BEDSIDE DIAGNOSIS OF CARDIAC IRREGULARITIES

The electrocardiogram is the method of choice for the diagnosis of disturbances in cardiac mechanism. This method of study is employed whenever feasible. However it is necessary that the attending physician [illegible] egularities of the [illegible] It is important [illegible] ie importance in [illegible] can be identified at the

1 Normal sinus rhythm
2 Sinus tachycardia
3 Sinus bradycardia
4 Sinus arrhythmia
5 Premature contractions
6 Paroxysmal tachycardia
7 Auricular flutter
8 Auricular fibrillation
9 Heart block
10 Pulsus alternans

Before undertaking this chapter the student should thoroughly acquaint himself with the mechanism of the heart beat and the transmission of impulses through the conduction tissue under normal and abnormal circumstances.

During the study of a patient for any type of complaint the cardiac evaluation should be approached in an orderly and organized manner. This approach in the practice of competent physicians is so commonplace that it becomes almost reflex in nature. If the physician is a careful observer and he should be trained to observe carefully, he begins checking the patient's cardiac mechanism when he first meets him, before the patient removes his clothes and especially while the history is being obtained. Constant observation of the rate and rhythm of the heart is maintained throu[illegible] The rate, rhythm or any [illegible] is simple observations whic[illegible] sical examination of a patien[illegible]

1 *Inspection* of the pulsations of peripheral vessels, both arteries and veins wherever visible, can be valuable. Precordial cardiac pulsations are also often informative.

2 *Palpation* of similar vessels and precordium during the palpatory examination frequently yields precise information of [illegible]

3 [illegible] the r[illegible]

4 [illegible] *ow sounds* heard durin[illegible] [illegible] blood pressure determination

It is the purpose of this chapter to show how much can be obtained by ordinary observation of simple phenomena which are usually passed over perfunctorily by the usual examiner. Where is the additional information obtained with more precise instruments, such as the phonocardiograph, the phlebograph and the electrocardiograph, is of extreme value and must not be underemphasized, these devices are adjuncts to diagnosis and do not replace the physician. A good clinician should be able to evaluate a patient entirely without their help.

NORMAL SINUS RHYTHM

The fundamental mechanism of the cardiac impulse is the *normal sinus rhythm*. This consists in the development and release of an impulse by the sino-auricular (SA) node at a rate of 60 to 90 times per minute. Each impulse released initiates a process of depolarization in the atrial musculature. The impulse stops momentarily at the atrioventricular (AV) node and is then released into the bundle of His, the two bundle branches and the network of Purkinje fibers from whence it is delivered to the ventricular musculature. At these points there is initiated a process of depolarization which spreads throughout the ventricles in an orderly manner, repeating itself just before each heart beat. This sequence of events occurs when the node is normal in function. It is followed by certain cardiovascular phenomena which can be readily observed by the attending physician in his study of the patient:

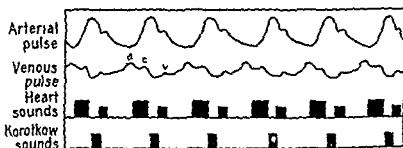


FIG 164.—*Normal sinus rhythm.* From above downward the *arterial pulse*, *jugular venous pulse*, *heart sounds* and *Korotkow sounds* are recorded simultaneously. In subsequent figures of this type the [illegible] are abbreviated [illegible]

1. Pulsations of superficial arteries and heart at the precordium
2. Pulsations of superficial veins
3. Heart sounds
4. Korotkow sounds, at least the sounds noted on recording a blood pressure

The *criteria for the diagnosis* of normal sinus rhythm are:

1. The rate is from 60 to 90 beats per minute.
2. The cardiac contractions are initiated by a normal pacemaker (SA node).

Establishment of the first criterion is obviously by inspection palpation and auscultation The proof that the SA node is the originating focus is dependent upon the fact that this node is under regulatory control If this can be demonstrated it must be concluded that such a rhythm is of a normal sinus mechanism (Fig 164) This can be accomplished at the bedside

(a) Increased vagal tone results from carotid sinus or ocular pressure which is manifested by retardation of the rate

(b) Deep breathing results in a similar phenomenon At the end of inspiration the rate decreases and becomes more rapid on deep expiration

(c) Exercise increases the rate gradually according to the degree of exertion The processes involved are too complex to discuss here

(d) Change in body posture alters the heart rate

In addition to these regulatory manifestations one can usually demonstrate a normal sequence of venous pulse waves Such a sequence indicates conclusively that an auricular contraction is being followed by a ventricular contraction

SINUS TACHYCARDIA

1 A regular rate of impulse formation exceeding 90 but rarely over 160 per minute is established automatically by inspection palpation and auscultation of the patient when he is *first seen* If adult cardiac rate exceeds 200 beats a minute it is unlikely to be tachycardia

2 Establishment of the SA node as the pacemaker in which impulse formation is essentially normal The establishment of the SA node as the pacemaker in a patient with tachycardia is the most difficult problem The criteria indicating the SA *node* as the pacemaker are

(1) The *history* is of great value With sinus tachycardia the cardiac rate gradually increases and subsides slowly as it returns to the normal resting rate In paroxysmal tachycardia the onset and cessation are *sudden* (*Fig* 165) This is also true of auricular flutter Careful *questioning* of the patient will yield the foregoing characteristics

(2) *Increase in vagal tone* by carotid sinus pressure or ocular pressure results in a gradual *reduction* in the cardiac rate The rate will slowly return to the previous level (*Fig* 166) In paroxysmal tachycardia or auricular flutter the ventricular rate may suddenly (within the space of one beat) decrease (Figs 165 and 167) In auricular flutter the rate promptly returns jerkily to its previous level

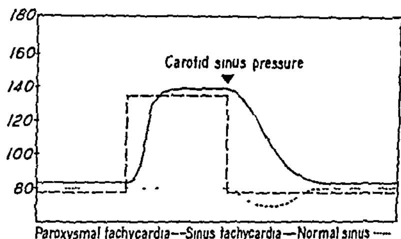


FIG 165 —The rate and degree of change in heart rate in *paroxysmal tachycardia sinus tachycardia* and *normal sinus rhythm* which characteristically occurs spontaneously and following pressure on the carotid sinuses Remember the parasympathetic innervation is only supraventricular in distribution Consult the text for detail

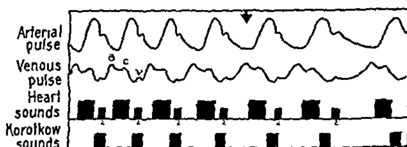


FIG 166 —*Gradual reduction in cardiac rate in sinus tachycardia following pressure* on the carotid sinuses

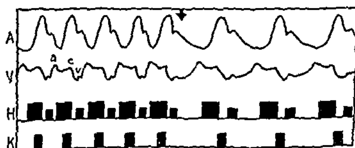


FIG 167 —Sudden reduction of cardiac rate within the interval of one cardiac cycle in paroxysmal supraventricular (auricular or nodal) tachycardia

(c) *Exercise* will gradually increase the cardiac rate as the exercise progresses. The increase is not particularly striking if the cardiac rate is already high, that is, around 140.

(d) *Deep breathing* varies vagal tone usually to a sufficient degree to produce a change in heart rate. At the end of deep inspiration the rate increases; it becomes less rapid near the end of expiration. A change in heart rate by respiration indicates the SA node to be the pacemaker of the heart. This is a practical test.

(e) *Change in body posture* changes the cardiac rate.

(f) The *jugular veins* will show *a* and *v* waves. Unfortunately, these are difficult to identify when the rate is rapid. With *training* this can be done, however. In uncomplicated sinus tachycardia an *a* wave should precede each *v* wave, indicating an equal number of auricular and ventricular contractions (Fig. 168).

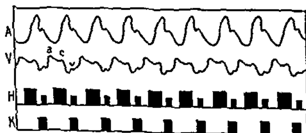


Fig. 168.—Sinus tachycardia: arterial pulse A, jugular pulse V, heart sounds H, and Korotkow sounds K. This lettering applies to all figures of this type.

(g) Ft olon ...

... emotionally upset ... develops tachy-
... anxiety

(i)

... nodal in
... does not constitute proof

reaction to

There is no specific *treatment* indicated for sinus tachycardia The cause should be removed when it can be established Satisfactory treatment of the cause such as congestive heart failure results in restoration of the heart rate to normal

SINUS BRADYCARDIA

Whenever the heart rate is less than 60, bradycardia exists regardless of the type of mechanism responsible for it If the SA node is the pacemaker and is responsible for the decreased rate, the mechanism is *sinus bradycardia* (Fig 169) The diagnostic approach is as described previously

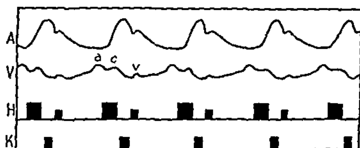


FIG 169 —*Sinus bradycardia*

The *diagnostic criteria* elicited at the bedside are

1 The *heart rate* is less than 60 but rarely less than 45 If it is lower than this it is likely to be complete AV block The rate is determined by inspection palpation and auscultation It is necessary to check the apical heart rate by listening to the heart sounds or palpating the apex beat for at times a patient will have a normal cardiac rate and a serious pulse deficit and therefore a slow pulse This often occurs with frequent premature contractions

2 A logical *cause* for sinus bradycardia may be present Sinus bradycardia may be caused by senility jaundice increase in parasympathetic (vagal) tone and congenital factors

3 The *history* is important Many people have congenitally slow heart rates Bradycardia which is of recent development such as that associated with complete heart block and Stokes Adams syndrome can be noted from the medical history

4 *Increase in vagal tone* by pressure on the carotid sinuses results in retardation of the heart rate an indication of SA control over the cardiac mechanism (Fig 170) Ocular pressure and the gag reflex may be tried if bilateral carotid sinus pressure fails In complete AV block an increase in vagal tone does not influence the ventricular rate as there is no parasympathetic innervation to the ventricles

5 *Exercise* increases the heart rate considerably when sinus bradycardia is present In complete or partial AV block there is little change in the ventricular rate with exercise This is an exceedingly simple and valuable test (Fig 171)

6 *Deep inspiration and expiration* should be tried as described for sinus tachycardia A proper response indicates that the SA node is the pacemaker

7 *Change in body posture* will change the heart rate

8 *Pulsations of the jugular vein* are of extreme value in identification of the SA node as the pacemaker When there is sinus bradycardia an auricular contraction precedes each ventricular one This results in an *a* wave and *c* wave for each cardiac cycle there being an equal number of each With complete AV block there are more *a* waves than *c* waves there being about twice as many Furthermore, the *a* and *c* waves have no constant time relationship to each other

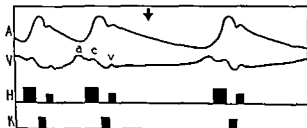


FIG 1 0 —*Sinus bradycardia* with further slowing following carotid sinus pressure evidence in support of the SA node as the pacemaker

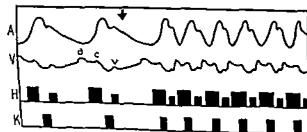


FIG 1 1 *Sinus bradycardia* with increase in heart rate following exercise another sign in support of the SA node as the pacemaker

these circumstances drugs such as ... episodes are encountered Under

SINUS ARRHYTHMIA

The rate with which the SA node forms impulses varies with the respiratory rate because of variations in vagal tone associated with (Hering-

Breuer reflex) breathing The heart rate declines near the end of inspiration and increases during the latter part of expiration The gradual waxing and waning of the heart rate with respiration is diagnostic of sinus arrhythmia (Fig 172) When this is noted it is almost certain that the SA node is the

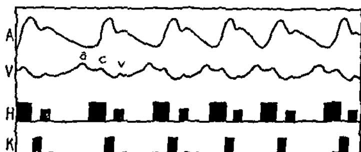


Fig 172 — Sinus arrhythmia

pacemaker of the heart The diagnosis may be established by demonstrating the a variations with respiration

Sinus arrhythmia is highly developed in children becoming less prominent with age It is relatively uncommon in the senile person *Deep breathing* will produce variations in heart rate regardless of age

Sinus arrhythmia has no *clinical significance per se* and requires no *treatment*

PREMATURE CONTRACTIONS

(Ectopic Beats Extrasystoles)

The term *premature contractions* or *premature beats* is preferred to the commonly employed terms *extrasystole* and *ectopic beats* which are satisfactory provided they are properly used It will be seen that all these terms indicate premature contractions but all premature contractions are not really extra contractions or ectopic in origin

By *definition* all beats or contractions that occur *prematurely* or earlier than expected are premature beats In fact this is the only criterion necessary to establish the diagnosis

Mechanism To understand the mechanism of premature beats or any type of *disturbance in cardiac mechanism one must be thoroughly acquainted* with the normal mechanism In the normal the impulse originates from a stimulus initiated at the SA node This stimulus starts a process of depolarization in the atrial musculature which is followed by contraction The impulse reaches the AV node as the depolarization process where it is delayed temporarily It is then released and passes down the bundle of His to the two bundle branches and thus into the Purkinje system where it is delivered to the subendocardial layer of ventricular musculature A process of depolarization in this muscle is initiated which migrates perpen-

dicularly toward the epicardial surface. It is followed by a contraction of the ventricles. This chain of events occurs in the heart of the resting person about 72 times each minute. Each one is followed by a fairly constant or predictable interval of time, varying only through the influence of respiration (sinus arrhythmia). The interval of time between each cycle of events is determined by the time required by the S A node to elaborate the initiating stimulus. In *sinus bradycardia* this time is long, whereas in *sinus tachycardia* it is relatively short. Under such a chain of events the chambers of the heart contract in normal sequence.

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upon the focus and time of initiation of the early impulse. This results in a disturbance in the rhythm of the heart beat (Fig. 173). The stimulus

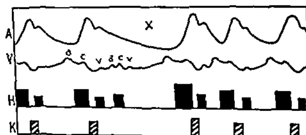


Fig. 173.—An *a ... premature* beat occurring early or prematurely upsetting th normal rhythm of the observed hemodynamic phenomena.

n

ectopic beats ... Therefore all *premature beats* are not

Premature beats are essentially of *three types*, depending upon the site of origin of the initiating stimulus.

1. Auricular premature contractions
2. Nodal (AV) ...
3. V

These

electrocardi... clinical ones ... ly especially the evident clinically or at the bedside, however. Certain differences are will be noted later under their individual discussions, which are not sufficiently reliable to establish an irrefutable diagnosis ...

...general physical examination

Breuer reflex) breathing The heart rate declines near the end of inspiration and increases during the latter part of expiration The gradual waxing and waning of the heart rate with respiration is diagnostic of sinus arrhythmia (Fig 172) When this is noted it is almost certain that the SA node is the

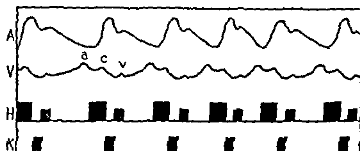


FIG 172 —*Sinus arrhythmia*

pacemaker of the heart The diagnosis may be established by demonstrating these variations with respiration

Sinus arrhythmia is highly developed in children becoming less prominent with age It is relatively uncommon in the senile person *Deep breathing* will produce variations in heart rate regardless of age

Sinus arrhythmia has no *clinical significance per se* and requires no *treatment*

PREMATURE CONTRACTIONS

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pre ure above the di stolic pressure in the aorta (Fig 175) Therefore, the aortic valves do not open and no blood is ejected from the ventricle When a contraction is too feeble to produce even a weak pul ation in a peripheral artery, the effect on the pul e is that of having a pul ation drop out often erroneou ly referred to by physicians as a dropped beat The lay expression dropped beat refers to the compensatory pause of all premature beats It is not a dropped beat for the heart actually contracts, as is evident from au cultation Inspection of the jugular venous pulsations may show evidence of the premature contraction (*a* wave) and/or *c* wave even though it is not manifested in the peripheral arteries An early impulse is more likely to produce a feeble contraction because the muscle is in partial refraction and therefore contracts only slightly, even though it does contract with its greatest available force

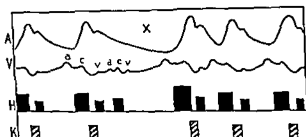


FIG 175—The *auricular pre at ure contraction* was too feeble to force blood out of the ventricles therefore there is no second heart sound no arterial pul e and no Korotkow sound note l while rec r ling the blood pressur

Furthermore early contraction results in less filling and thus a weak contraction The earlier the appearance of the premature beat the more feeble is the muscular contraction

The two types of pul atile manifestations described are found not only on *inspection* of pulsating vessels or *clothing* pushed by these vessels but are also observed on *palpation* of the peripheral arteries

The same two types of manifestations of premature beats are also obtained by *auscultatory* methods Evidences of premature contractions are obtained during the *routine measurement of arterial* [illegible]

(Fig 176) [illegible]

that is they [illegible]

greater than [illegible] arterial pressures Listening to the Korotkow sounds will reveal a fundamental rhythm for the sound When a premature contraction occurs which is strong enough to force a pulse wave down the artery a *prematurely appearing less intense sound* will occur followed by a compensatory pause and then one of the regularly appearing sounds which [illegible]

are in progress This allows for a fairly prolonged period of observation, which is often necessary when the premature beats are infrequent

Observation of the pulsating superficial vessels of the body will reveal a regular rhythm When a premature contraction appears, either of two things can occur

1 There is an earlier appearance of a pulsation than would be expected by the previous rhythm The pulsation seen is almost invariably *weaker* than the previous ones This beat is weak because the ventricles are partially refractory and incompletely filled (Starling's law of the heart) and therefore contract rather feebly Following the premature beat there is a compensatory pause In general this is *incomplete* in auricular premature beats and nodal premature beats with retrograde conduction and *complete* in ventricular premature beats and nodal ones without retrograde conduction The first pulsation following the compensatory pause is

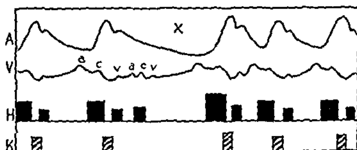


Fig 174 —A premature contraction is weaker than those occurring normally The earlier it occurs in the cardiac cycle the more feeble it is The contraction following the compensatory pause is forceful because of the greater period for filling of the ventricle and therefore more ventricular filling (Consult the text for details) The hemodynamic phenomena occurring with the feeble contractions are feeble and those associated with the strong contraction are stronger In this instance the premature contraction was not strong enough to produce an arterial pulse (thus second sound and Korotkow sound absent)

usually more forceful than the regularly appearing ones The large pulsations occur because the compensatory pause allows longer and therefore more complete ventricular filling therefore a greater stroke volume and force occur at the time of the next ejection (Fig 174) With more stretching there is greater force of contractions (Starling's law of the heart) A premature contraction that is stronger and later in appearance is likely to be one that is strong enough to open the aortic valves and force a column of blood and a pulse wave, with all its manifestations, to the periphery

2 The other manifestation may consist in a basic cardiac rhythm with the premature contraction failing to produce a peripheral pulsation, thus an *interval without any pulsations* The next pulsation is a strong one The duration of this interval varies with the type of compensatory pause The premature contraction fails to produce a peripheral pulsation when the ventricular contractions are too feeble to raise the intraventricular

is a pause and then the relatively loud sounds of the first contraction of the regular rhythm Obviously when the contractions of the regular rhythm are also associated with the third and fourth heart sounds the grouping of the heart sounds will vary accordingly It is not difficult to predict the groupings

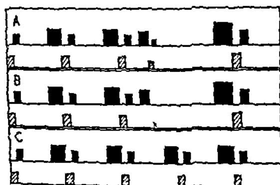


Fig 178 —Th ...

Part ...
the firs ...
mature beat is ...
sound also A f ...



Fig 179 A premature ...

At times a *premature* contraction may be too feeble to open the aortic valve but strong enough to open the pulmon... Under such circumstan... beat ... with the premature area ... the aortic valvular valvular area ... transmitted feebly from the pulmonary

Korotkow sounds for a moment followed by a sound of the fundamental rhythm but of greater intensity (Fig 177)

Auscultation of the heart is the most certain method of detecting premature contractions. This is done by observing the heart sounds. As indicated previously the heart sounds are produced by contraction of the heart. Therefore if the heart contracts even feebly and prematurely sounds will be produced. It follows then that the premature appearance of heart sounds indicates a premature contraction. Listening to the heart sounds will reveal a fundamental rhythm of appearance of the heart sounds



Fig 176 A premature contraction which was sufficiently strong to force blood out of the ventricles thereby resulting in a second heart sound and Korotkow sound



Fig 177 — A premature beat too feeble to force blood out of the ventricles and therefore resulting in absence of a Korotkow sound and arterial pulse

When a premature beat occurs there is premature occurrence of heart sounds. If the contraction is strong enough to open the aortic and pulmonic valves when the ventricles go into systole followed by closure when the ventricles go into diastole there will be a premature occurrence of at least the *first* and *second* heart sounds or a grouping of *four* heart sounds (two of the fundamental rhythm and two of the premature beat). The latter are less *intense than the regularly occurring ones* (Fig 178). Following the prematurely appearing sounds is a compensatory pause. The first sounds of the fundamental rhythm which appear after the pause are more *intense* than the other sounds. When the premature contraction is too feeble to open the aortic and pulmonic valves only a *first heart* sound is heard (Fig 177). The first sound of the premature contraction is followed

Premature beats may alternate with regular beats to produce *bigeminy* (Fig 181) This type of bigeminy is characterized by

1. Pairing of a strong beat and an early appearing weak beat a pause and then a strong beat, weak beat, and a pause etc (Fig 181) These types of pulsations are noted in the peripheral vessels by inspection palpation and auscultation of Korotkow sounds
2. The grouping of the heart sounds into groups of three or four followed by a pause The sounds associated with the premature beat are fainter than those of the regular contraction (Fig 181)

Bigeminy may follow partial (3 2) AV block (Fig 182) In this instance



Fig 182 —*Bigeminy* produced by a 3 2 AV block Every third impulse from the atria fails to penetrate the AV node thus resulting in asystole of the ventricles This pause produces grouping of the ventricular contractions and arterial pulsations into pairs

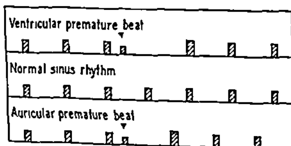


Fig 183
fails to upse
The norma

the atria contract in a regular manner The ventricles fail to respond to every third impulse initiated by the SA node There

an auricular sound which is present at a time expected according to the fundamental cardiac rhythm

It is sometimes difficult to differentiate between *premature beats of auricular and those of ventricular or a n* It is important to make this ricular origin are usually of after myocardial infarction

It must also be remembered that an auricular premature contraction is not always followed by a response of the ventricles. For example, an early premature auricular beat may result in the appearance of the impulse at the AV node while it is still in the refractory period and is therefore unable to respond and transmit the impulse to the ventricles. Under such a circumstance there may be no audible sound or there may be an audible auricular sound appearing soon after the second heart sound (Fig. 180).



Fig. 180.—When an auricular premature beat occurs early, the associated impulse may reach the AV node when it is in a refractory state too great to transmit the impulse to the ventricles. The impulse is thus blocked and the ventricle remains silent, and the hemodynamics associated with ventricular activity are absent. The auricular premature contraction often produces a faint audible sound (indicated by the arrow) which has the auditory effects of an echo.

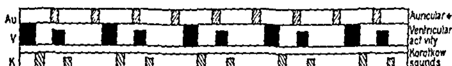


Fig. 181.—Illustration showing from above downward (1) *auricular activity* (Au), (2) *ventricular activity* (V), and (3) *Korotkow sounds* (K) in *coupling* and *pulsus bigeminus* due to ventricular premature beats occurring after each normal one. Consult text for details. This labeling scheme applies to all figures of this type in contrast to Figure 164.

When premature contractions occur only occasionally, there is no difficulty in *recognizing* them. When they occur at frequent intervals and in fairly rapid succession they may be difficult to differentiate from auricular fibrillation. A fairly good test in differential diagnosis is the response to exercise. The premature beats are reduced in number or completely disappear with exercise as the fundamental rate increases. In some instances, however, especially when there is organic heart disease, the premature beats are increased in number by exercise. Auricular fibrillation, on the other hand, becomes even more irregular with exercise.

' (1)
i the
vity

of murmurs (Fig 185) The influences should be obvious to the student by now The student should work out for himself some of the changes in the appearance of murmurs produced by premature beats

When there are *interpolated* premature beats, that is, a premature beat sandwiched in between two regularly appearing beats, a true *extrasystole* or extra beat exists (Fig 186) The main differences from the usual type of premature beat are

1 There is no or a short, compensatory pause and
2 The first regular contraction following the interpolated premature beat is usually more feeble than the regular cardiac contractions



Fig 185 – In a patient with heart murmurs, a premature contraction produces an early appearance of the murmur as shown above for an apical systolic murmur The intensity of the murmur is reduced because the force of the premature contraction is less than that of the regularly occurring contractions The murmur extends throughout systole in this illustration and fuses with the second sound

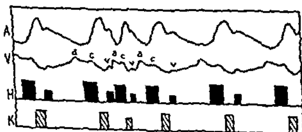


Fig 186 Illustration of an interpolated premature contraction producing an interpolation of the arterial and jugular pulses heart sounds and Korotkow sounds

The cause ... they occur fr... known that more likely t... They are infections or ... are state whether it is ... ety fatigue, mature be...

Prem... frequen... should ... considered as strong evidence of myocardial disease ... ental in origin They then ... It is true,

There are certain important characteristics of *ventricular premature contractions*

1 The *fundamental rhythm* is usually not disturbed This is observed by the appearance on time of the regular contractions (Fig 183) The timing of the regular beats is accomplished by tapping one's foot with each pulse beat or first heart sound and continuing this through the period when the premature beat appears The regular beat occurring after the compensatory pause will be found to be synchronous with the tapping if the premature contraction is of ventricular origin since the compensatory pause was *complete* and the SA node continued to initiate impulses at the regular rhythm If there is an *auricular premature* contraction the beat

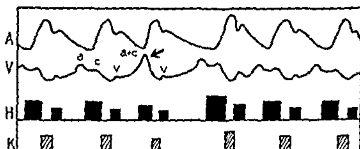


Fig 184 —When there is a late ventricular premature contraction occurring so as to result in simultaneous contraction of the atria and ventricles a strong pulsation of the jugular vein is produced Consult the text for details

occurring after the compensatory pause appears earlier than expected by the rhythm tapped by the observer's foot (Fig 183) This is true because the compensatory pause after the auricular premature beat is *incomplete*

2 While the *jugular pulsations* usually do not aid in differentiating auricular from ventricular premature beats by the appearance of the *a* waves on time in ventricular premature beats and prematurely with auricular premature beats (the *a* waves are often difficult to identify in premature contractions) such a method of differentiation should be sought as it may *be useful at times* *Of more importance however* is the appearance of a large *a* wave when a ventricular premature beat coincides with an *a* wave Under such circumstances the atria and ventricles contract simultaneously *The blood from the right atrium is unable to enter the* right ventricle through the closed AV valves held closed by the high intraventricular pressure and therefore regurgitates into or enters the venae cavae producing a *large or jerking type of pulsation* in the jugular vein (Fig 184) The first heart sound may also be exaggerated when a ventricular premature contraction occurs simultaneously with atrial contraction (Fig 184) Under such circumstances the auricular sound exaggerates the first sound of the premature ventricular contraction

It should be remembered that premature beats not only have their influence on the normal heart sounds but also produce a new grouping

The *cause* of paroxysmal tachycardia is not known It occurs in normal people as well as in those with heart disease Certain factors are known to predispose to the irregularity It is known to occur with fatigue anxiety states infections organic heart disease digestive disturbances malnutrition or almost any disturbance in mental or physical health

It occurs most often between the *ages* of twenty and thirty but may occur at any age It is much more common in men than women

Classification of Paroxysmal Tachycardia —Paroxysmal tachycardia is classified according to the site of the pacemaker initiating the impulse

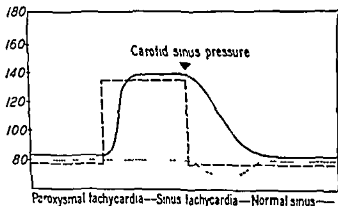


Fig 18 —Th
s n
and
t o

1 Auricular
2 Junctional or nodal (AV)
3 Ventricular

This differentiation is usually made only with the electrocardiograph *Paroxysmal ventricular tachycardia* for practical ol

flutter

Recognition at the Bedside of Paroxysmal Tachycardia in General —The observations of the manifestations of parox

al are

Auscultation of the heart sounds murmurs and Korotkow sounds Paroxysmal tachycardia presents the following characteristics which can be elicited by palpation percussion and auscultation (Fig 187)

however that the cardiac disease associated with premature beats is often reversible and of little or no significance

Treatment —The treatment of premature beats consists essentially in eliminating the precipitating and aggravating causes when possible Drugs are resorted to only when general hygienic measures fail If there is underlying heart disease proper management of this usually results in disappearance of the premature contractions In brief the therapeutic procedures are essentially as follows

1. *Rest* The patient should be advised to sleep longer hours take frequent naps or rest reclining frequently *Hard physical work* should be avoided temporarily *Mental* rest should be emphasized If there is any *anxiety state*, it should be properly managed
2. *Smoking* should be prohibited
3. *Caffeine* drinks should be avoided
4. *Alcohol* should not be used
5. The *diet* should be adequate and nourishing and general hygienic measures including bowel function avoidance of overeating and the like should be instituted
6. The underlying *cardiac disease* should be managed properly when present
7. *Drugs* are to be employed as a last resort Among those of value are
   - (a) *Sedatives should be given to relieve anxiety and insomnia when* the foregoing measures fail Phenobarbital (1 grain or 60 mg two or three times a day or as indicated) or sodium bromide (15 grains or 1 gram two or three times daily in the absence of congestive heart failure) may be used Do not use these drugs too frequently
   - (b) *Quinidine sulfate* (6 grains or 0.4 gram every two or three hours) should be used if the discomfort produced by the premature beats is annoying Always make sure the patient is not sensitive to quinine or its derivatives Quinidine therapy should be employed for a few days or two to three weeks then discontinued Usually the premature beats fail to return

Paroxysmal Tachycardia

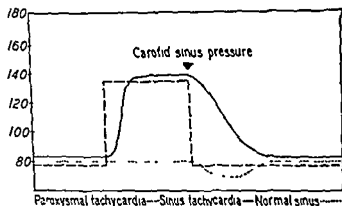
Paroxysmal tachycardia consists of paroxysms of rapid regularly occurring contractions which begin suddenly and end suddenly

The *mechanism* responsible for the paroxysms of tachycardia is not well known It is *generally thought* to be due to an *ectopic focus* in the heart which releases impulses in rapid succession and with absolute regularity Some observers suggest that it may be due to a circus movement *in which* the circuit includes the AV or SA *nodes* As the impulse passes through either of these nodes an impulse from the node initiates a wave of depolarization those originating from the SA node result *in* auricular tachycardia whereas those from the AV *node result in nodal tachycardia* Regardless of the fundamental physiologic mechanism the clinical manifestations of the disturbance are fairly characteristic

The *cause* of paroxysmal tachycardia is not known It occurs in normal ... as well as in those with heart disease Certain factors are known

It occurs most often between the *ages* of twenty and thirty but it ... occur at any age It is much more common in men than women

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2 Junctional or nodal (AV)
3 Ventricular

This differentiation is usually made only with the electrocardiograph

Paroxysmal ventricular tachycardia for practical ... urposes, is ... If there are ... not clini- ... be a theoretic difference between paroxysmal ventricular tachycardia and ventricular flutter

Recognition at the Bedside of Paroxysmal Tachycardia in General —The observations of the manifestations of parox ... al are

... peripheral vessels and the cardiac impulses

3

Korotkow sounds

... the following characteristics which can be elicited by palpation, percussion and auscultation (Fig 187)

1 The *rate* rarely exceeds 180 per minute in the adult. It is usually around 160 but may be more rapid or slower.
2 The *rhythm* is absolutely regular, one cycle varying from the other by not more than a hundredth of a second.
3 The *onset* and *end* of the paroxysm is *absolutely abrupt*, that is, it starts and ends within one cardiac cycle. This may be elicited by careful questioning of the patient.
4 Variations in *parasympathetic or vagal tone* have either of two influences on the heart rate (Fig. 188).

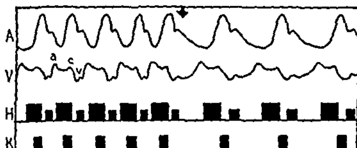


Fig. 188.—*Paroxysmal tachycardia* with sudden retardation (within one cardiac cycle) of the heart rate following carotid sinus pressure.

(*a*) It does not change at all.
(*b*) It changes abruptly, often stopping the paroxysm.
Vagal tone can be increased by pressing on the carotid sinuses (carotid sinus reflex) or eyeballs (oculocardiac reflex).

5 A change from the supine to the upright position or *vice versa* does not change the heart rate as it does when the normal pacemaker or SA node controls the mechanism. Rest, change in posture, and exercise will change the rate of sinus tachycardia.

Paroxysmal tachycardia may produce *symptoms*. Among the most common are:

1 *Anxiety* or apprehension, and at times panic in a patient who is not aware of the significance of the tachycardia.
2 Palpitation.
3 Dyspnea.
4 Dyspepsia.
5 Precordial discomfort or even anginal pain.
6 Weakness, fatiguability, vertigo, lethargy, and other manifestations of impaired cardiac output.
7 Manifestations of left and right ventricular congestive heart failure.
8 Delirium, coma, and death may be the terminal manifestations of a ...

ventricular paroxys-
cardiogram. Should
m
carotid sinus pressure stop a paroxysm, it can be stated definitely that the

tachycardia was not ventricular in origin for there is no parasympathetic innervation to the ventricles

Paroxysmal auricular tachycardia may be associated with a variable AV block This results in irregular ventricular contraction (Fig 189) which is often difficult to differentiate from auricular fibrillation If there are *a* waves in the jugular pulse or auricular sounds are heard, then auricular fibrillation does not exist (Fig 190)



Fig 189 *Paroxysmal tachycardia* with a *variable AV block* showing the regularity of auricular activity and irregularity of ventricular contraction and the peripheral radial pulse



Fig 190 —*Auricular fibrillation* with extreme variation in the time of appearance and force of the ventricular contractions and the peripheral arterial pulse This should be compared with figure 189 Consult the text for differential diagnosis

Paroxysmal tachycardia is difficult to *differentiate* from auricular flutter The main differences are

1 Auricular flutter has a more rapid auricular rate (240 or more) and as a rule a ventricular rate half that of the auricular one due to a 2 1 AV block This may be determined at times by observing that the *a* waves in the jugular pulse occur twice as frequently as the heart beats or radial pulsations although it is doubtful that the *a* waves can be identified and counted at such a rate
2 Pressure on the carotid sinuses stops or retards auricular flutter for only a short time The original rate is resumed jerkily just as soon as or even before pressure is released

Occasional attacks of paroxysmal tachycardia have no *clinical significance* in themselves unless they persist long enough to produce congestive heart failure or death Ventricular tachycardia should not be considered normal but auricular and nodal tachycardia are frequently present in normal persons The underlying cardiac state determines the significance of the paroxysms of tachycardia

Treatment —The treatment of paroxysmal tachycardia is about the same as that for premature contractions The general measures of rest hygiene abstinence from tobacco alcohol or caffeine drinks proper sedation etc should be emphasized Any underlying *cardiac state* should

1 The *rate* rarely exceeds 180 per minute in the adult It is usually around 160 but may be more rapid or slower
2 The *rhythm* is absolutely regular, one cycle varying from the other by not more than a hundredth of a second
3 The *onset* and *end* of the paroxysm is *absolutely abrupt* that is it starts and ends within one cardiac cycle This may be elicited by careful questioning of the patient
4 Variations in *parasympathetic or vagal tone* have either of two influences on the heart rate (Fig 188)

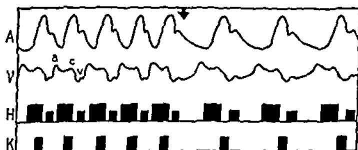


Fig 188 —*Paroxysmal tachycardia* with sudden retardation (within one cardiac cycle) of the heart rate following carotid sinus pressure

(*a*) *It does not change at all*
(*b*) It changes abruptly often stopping the paroxysm
Vagal tone can be increased by pressing on the carotid sinuses *(carotid sinus reflex) or eyeballs (oculocardiac reflex)*

5 A change from the supine to the upright position or *vice versa* does not change the heart rate as it does when the normal pacemaker or *SA node controls the mechanism Rest change in posture* and exercise will change the rate of sinus tachycardia

Paroxysmal tachycardia may produce *symptoms* Among the most common are

1 *Anxiety* or apprehension and at times panic in a patient who is not aware of the significance of the tachycardia
2 *Palpitation*
3 Dyspnea
4 Dyspepsia
5 *nginal pain*
6 lethargy and other manifestations
7 ventricular congestive heart failure
8 be the terminal manifestations of a prolonged episode

It is *impossible to differentiate auricular nodal and ventricular* paroxysmal tachycardia with certainty without an electrocardiogram *Should carotid sinus pressure stop a paroxysm it can be stated definitely that the*

pulse is delivered to the AV node The node is not able to respond to each impulse reaching it so that a 2 1 3 1 or variable block occurs thus the ventricular rate is slower than the auricular rate The ventricular rate is usually one-half to one-third the auricular rate Auricular flutter is rarely found in normal hearts It is most often associated with hyperthyroidism mitral stenosis arteriosclerosis and hypertension The highest total incidence is in arteriosclerosis whereas the highest percentage incidence is found in hyperthyroidism and mitral stenosis It occurs at any age and is four times more frequent in *men* than in women Infections tobacco caffeine drinks and mental and physical fatigue predispose to auricular flutter

Bedside Diagnosis The recognition of auricular flutter is sometimes possible although the mechanism cannot always be identified with certainty It is recognized clinically by the methods described for premature contractions that is *inspection* and *palpation* of pulsating vessels and the precordium and *auscultation* of the heart and Korotkow sounds



Fig 191 —*Auricular flutter* showing the arterial and jugular pulses heart sounds and Korotkow sound

Clinical characteristics

1 A careful history will re...
the patient

2 If the paro...
does not v... ...rest or *exercise* it is almost certainly auricular flutter The auricular rate may be rapid for such long periods of time without undue cardiac embarrassment because there ...
may be over 300

3 ...
of 300 or more and *v* waves or a ventricular rate of 150 is strongly in favor of auricular flutter

4 ...

5 ...

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medium of
13

be promptly and adequately treated. Any underlying systemic disease known to produce paroxysmal tachycardia, such as hyperthyroidism, should be alleviated when possible. Should the paroxysms occur at frequent intervals or last for prolonged periods of time, more *specific drugs* should be employed.

1. *Carotid sinus or ocular pressure* should be tried before any drugs are employed. An increase in vagal tone is usually sufficient to arrest a paroxysm of tachycardia. Touching the pharynx to produce gagging may also be tried to increase vagal tone. Vomiting is particularly unpleasant during the paroxysms, however.
2. *Quinidine sulfate* in adequate doses must be tried. For the usual patient 6 grains (0.4 gram) administered orally every two or three hours is sufficient. The dose is then reduced to 6 grains (0.4 gram) three or four times daily. This may be continued for several days or a few weeks if necessary. For an episode that has been prolonged and is *beginning to produce signs of congestive heart failure* the dose should be increased to 6 grains (0.4 gram) every hour or two until the paroxysm stops or signs of intoxication develop. It is necessary to run the risk of intoxication when a patient is developing failure, *for death will certainly ensue if the tachycardia persists. Fear of the use of quinidine is far greater than is warranted by* clinical experience. Once a paroxysm has stopped, the drug should *be continued for several days in doses of 6 grains (0.4 gram) every* three or four hours to prevent recurrence. Quinidine is the drug of choice. It may be administered intravenously in doses of 5 to 6 *grains (0.3 to 0.4 gram) every three hours if oral administration* fails or is contraindicated.
3. *Acetyl beta methyl choline* in doses of 20 to 30 mg. is recommended and used by some clinicians with success. The dose may be repeated in two or three hours if necessary. If large doses of quinidine fail, acetyl beta methyl choline is unlikely to produce results.
4. *Digitalis* is employed by many clinicians to arrest the paroxysms even if congestive heart failure is not present. It is indicated if congestive heart failure is present and should be administered as outlined for this disease. Digitalis is more likely to be effective if organic heart disease is present. It should be used cautiously, if at all, in the presence of paroxysmal ventricular tachycardia, as there is a possibility that the tachycardia will be converted to ventricular *fibrillation*.

Auricular Flutter

Auricular flutter is a disturbance in cardiac mechanism which is associated with an absolutely regular atrial rate of 240 beats or more per minute. It is contended by some to be due to a regular circus movement in the atrium, whereas recent studies suggest that the impulses arise from a single ectopic focus. Each time a wave of activity is completed, another impulse is initiated which results in an auricular contraction, and an im-

Auricular fibrillation is considered to be due to numerous circus movements as "islets" of contracting auricular muscle which may be in any stage of depolarization or repolarization. As a result the atria remain distended (in diastole) and merely quiver, undulate, twitch or fibrillate but never contract completely to empty themselves of blood. The AV node is therefore receiving many impulses of extremely variable intensity and timing. Those that reach the node when it is refractory or relatively refractory fail to pass through to the ventricles. The weaker impulses particularly fail to initiate an AV impulse. Because of the irregular conduction of impulses to the ventricles, the ventricular rate is irregular. Furthermore, the earlier the impulse reaches the ventricular musculature, the greater will be the state of refraction and the greater the refraction the less force of contraction. The variations in the force of contraction result in an



Fig. 133.—The arterial pulse, jugular pulse, heart s[illegible]

identical variation in the force of the pulsations in the peripheral vessels, intensity of the heart sounds and intensity of Korotkow sounds. There is therefore an absolute irregularity in ventricular activity and irregularity in the rate and force of the arterial pulse. Fibrillation may occur in short *paroxysms* of a few seconds, minutes, hours or days or may be *chronic* and *persist* for many years.

The cause of auricular fibrillation is not clear. It varies much like auricular flutter in its incidence, occurring especially in hyperthyroidism, mitral stenosis, arteriosclerosis and hypertension. It occurs most often in *older* people but it can occur at any age. *It is more common in men* than in women. It is frequently precipitated by infections, congestive heart failure, myocardial infarction, tob[illegible]

…upon two factors

6 Exercise will cause the ventricular rate to increase and become regular if variable AV block is present

The *differential diagnosis* is usually difficult It is most often confused with simple paroxysmal tachycardia The characteristics listed previously for the two mechanisms serve to differentiate them

*Auricular flutter with irregular AV block is often confused with auricular fibrillation The differentiation is simple A mild degree of exercise in*creases the ventricular rate and makes it regular a characteristic never present in auricular fibrillation (Fig 192)

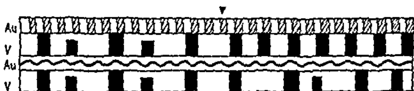


Fig 192 —*Comparison of auricular flutter with a variable AV block with auricular fibrillation* Wedge indicates onset of change from emotional excitement or exercise Consult the text for detail

Auricular flutter with a variable AV block may be confused with multiple and rapidly occurring premature contractions Exercise usually changes both mechanisms to a regular rhythm but *in auricular flutter less exercise brings about the change and the increase in rate tends to be out of pro*portion to the degree of exercise With multiple premature beats the irregularity may be increased at times or the mechanism may return to a regular rhythm with relatively little increase in rate

The *symptoms* produced by auricular flutter are essentially the same as those described previously for paroxysmal tachycardia Syncope may be *a prominent feature of the symptom complex*

Auricular flutter is a sign of heart disease the underlying cardiac state determines the significance of the heart disease rather than the flutter itself

Treatment —The treatment of paroxysmal auricular flutter is the same as that described for paroxysmal tachycardia except that acetyl beta methyl choline and measures to increase vagal tone are not employed therapeutically *If the auricular flutter tends to be chronic and fails to* respond to the other measures then digitalis should be administered in the hope of converting the flutter to fibrillation Once this has been achieved the ventricular rate *can be maintained at about 70 to 80* a rate which is not deleterious The mechanism frequently reverts to normal following digitalis therapy

Auricular Fibrillation

Auricular fibrillation is a disturbance in mechanism due to many separate circus movements *or numerous foci initiating impulses* in the atria which results in incomplete isolated auricular contractions at a rate of about 500 per minute The ventricular rate *is variable* and *the rhythm and force of* contractions are *absolutely irregular*

Auricular fibrillation is considered to be due to numerous circus movements as islets of contracting auricular muscle which may be in any stage of depolarization or repolarization. As a result the atria remain distended (in diastole) and merely quiver, undulate, twitch or fibrillate but never contract completely to empty themselves of blood. The AV node is therefore receiving many impulses of extremely variable intensity and timing. Those that reach the node when it is refractory or relatively refractory fail to pass through to the ventricles. The weaker impulses particularly fail to initiate an AV impulse. Because of the irregular conduction of impulses to the ventricles the ventricular rate is irregular. Furthermore the earlier the impulse reaches the ventricular musculature the greater will be the state of refraction and the greater the refraction the less force of contraction. The variations in the force of contraction result in an



FIG 193 *The arterial pulse, jugular pulse, heart sound, murmurs of Korotkow sounds in mitral stenosis with a normal sinus mechanism changing to auricular fibrillation.* The X marks the absence of *a* waves and late diastolic crescendo murmurs with the fibrillation because of the lack of organized and complete contraction of the atria.

identical variation in the force of the pulsations in the peripheral vessels, intensity of the heart sounds and intensity of Korotkow sounds. There is therefore an absolute irregularity in ventricular activity and irregularity in the rate and force of the arterial pulse. Fibrillation may occur in *short paroxysms* of a few seconds, minutes, hours or days or may be *chronic* and persist for many years.

The cause of auricular fibrillation is not clear. It varies much like auricular flutter in its incidence, occurring especially in hyperthyroidism, mitral stenosis, arteriosclerosis and hypertension. It occurs most often in *older* people but it can occur at any age. It is more common in *men* than in women. It is frequently *precipitated* by infections, congestive heart failure, myocardial infarction, tobacco, coffee ...
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... diagnosis of auricular fibrillation at the bedside ...
the method of ...
The n

1 Virtual paralysis of the atria
2 Disturbance in ventricular contraction

Because of *failure of the atria to contract,* there are no *a* waves in the pulsations of the jugular veins no auricular or fourth heart sound and no murmur which is dependent upon auricular systole (Fig 195) The latter two are especially important diagnostically if either or both were present before the onset of the irregularity and are subsequently found to be absent Furthermore, *if a fourth or auricular heart sound or a late diastolic crescendo murmur is heard auricular fibrillation cannot exist If a waves are identified auricular fibrillation cannot exist* It is obvious therefore that

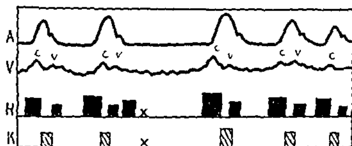


Fig 194 —Illustration of variations in rhythm and force of the arterial and jugular pulses and heart sounds and Korotkow sounds in *auricular fibrillation*



Fig 195 —*Auricular fibrillation* with a pulse deficit due to the feeble ventricular contractions being unable to eject blood into the aorta from the left ventricle The X marks the absence of arterial pulsations

the auricular paralysis is of considerable or of greatest diagnostic importance A *concerted search for the presence or absence of auricular activity* should be made

The nature of the *ventricular contractions* is usually sufficient to establish the diagnosis of auricular fibrillation It is however not nearly as certain as the demonstration of auricular paralysis As indicated previously the ventricular contractions are (1) irregular in timing or rhythm and (2) irregular in force These changes manifest themselves in the pulsations in the peripheral vessels heart sounds and Korotkow sounds (Fig 194) These manifestations are detected by inspecting and palpating the peripheral arteries jugular veins and precordial pulsations due to the heart *per se* Auscultation of the heart reveals the *variations in timing and intensity* of the sounds The same is true with the Korotkow sounds when the blood pressure is being recorded

The *peripheral arterial pulsations* show the following (Fig 195) The pulsations appear at variable periods of time and they vary in intensity The closer one occurs to a previous one the more feeble the pulsation (Fig 195) Some of the ventricular contractions are too feeble even to force blood through the aortic valve thus resulting in a ventricular contraction without an associated arterial pulsation Such an occurrence is known as a *pulse deficit* It is for this reason that a determination of the arterial pulse rate is no index of the rate of ventricular contractions and why the *apical heart rate must be counted* Furthermore a ventricular contraction that is too feeble to force blood into the arteries not only is useless but fatigues and overworks the heart unnecessarily The greater the pulse deficit the less efficient is the circulation

The jugular pulsations show irregularly occurring *c* and *v* waves but no *a* waves (Fig 196) The extremely feeble ventricular contractions may fail to produce *v* waves in the jugular pulse



Fig 196 — *Auricular fibrillation* with irregularly appearing *c* and *v* waves without any *a* waves

The blood pressure determination presents evidence of auricular fibrillation in that the Korotkow sounds vary considerably in intensity and timing (Fig 196) Because of the great variations in intensity of the force with which the ventricles pump blood to the periphery, the blood pressure cannot be recorded accurately and must be estimated For example during recording of the blood pressure the pressure within the pneumatic cuff is brought to a level sufficient to exclude all Korotkow sounds below the cuff By a gradual lowering of the pressure within the cuff a pressure level is reached where only an occasional strong ventricular contraction forces blood through and a sound is produced W...
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reached ... appear as the fifth phase is

Auscultation of the heart reveals wide variations in intensity and timing of the sounds When the ventricular contr...
the ...

from the hemodynamics concerned (Fig 198) There will be no auricular heart sound or late diastolic crescendo murmur because of the auricular paralysis

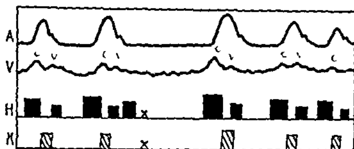


Fig 197 —*Auricular fibrillation showing the variations in intensity and timing of the arterial and jugular pulses heart sounds and Korotkow sound.* These phenomena are more feeble when ventricular contractions occur early and more intense when there is a long interval between contractions Such occurrences are physiologically obvious



Fig 198 —*Normal sinus mechanism changed suddenly to auricular fibrillation* with variations in timing and intensities of murmurs in mitral insufficiency and stenosis The late diastolic crescendo murmur disappears with the onset of auricular fibrillation

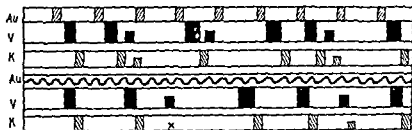


Fig 199 —*Comparison of normal sinus rhythm with frequent ventricular premature contractions with auricular fibrillation* Consult the text for detail

Auricular fibrillation may be confused with

1 Multiple and frequent premature beats
2 Auricular flutter with a variable AV block
3 Paroxysmal tachycardia with a variable AV block

Differentiation between auricular fibrillation and frequent premature contractions (Fig 199)

(a) Following exercise *premature beats* usually disappear and the rhythm becomes regular, whereas it usually increases the irregularity in auricular

fibrillation (*b*) If an irregularity exists at a rate of 120 or more, then it is most likely to be auricular fibrillation (*c*) If auricular or fourth sounds are heard auricular fibrillation does not exist and premature contractions are probably present (*d*) The presence of a presystolic crescendo murmur at the apex eliminates auricular fibrillation as the mechanistic disorder (*e*) The presence of *a* waves in the jugular pulse rules out auricular fibrillation

Differentiation between auricular fibrillation and auricular flutter with variable AV block (Fig 200)

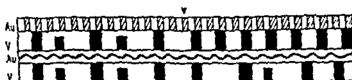


Fig 200 *Comparison of auricular flutter with a variable AV block with auricular fibrillation* Consult the text for details

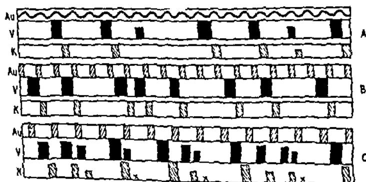


Fig 201 — *Comparison of auricular fibrillation A with sinus tachycardia with a variable AV block B with normal sinus rhythm and frequent ventricular premature contractions, C*

1 After exercise the auricular flutter with variable block becomes regular as the AV conduction is made constant Auricular fibrillation is made more irregular by exercise

2 The presence of *a* waves auricular or fourth heart sound or a presystolic crescendo murmur eliminates auricular fibrillation as these cannot exist with auricular paralysis

3 D

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from the hemodynamics concerned (Fig 198) There will be no auricular heart sound or late diastolic crescendo murmur because of the auricular paralysis

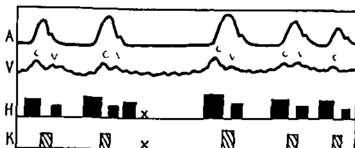


Fig 197 —*Auricular fibrillation* showing the variations in intensity and timing of the arterial and jugular pulses heart sounds and Korotkow sounds The c phenomena are more feeble when ventricular contractions occur early and more intense when there is a long interval between contractions Such occurrences are physiologically obvious



Fig 198 —*Normal sinus mechanism changed suddenly to auricular fibrillation* with variations in timing and intensities of murmurs in mitral insufficiency and stenosis The late diastolic crescendo murmur disappears with the onset of auricular fibrillation

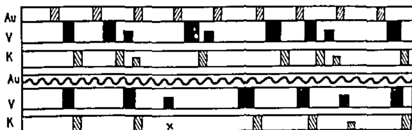


Fig 199 —*Comparison of normal sinus rhythm with frequent ventricular premature contractions with auricular fibrillation* Consult the text for detail

Auricular fibrillation may be confused with
1 Multiple and frequent premature beats
2 Auricular flutter with a variable AV block
3 Paroxysmal tachycardia with a variable AV block

Differentiation between auricular fibrillation and frequent premature contractions (Fig 199)

(*a*) Following exercise *premature beats* usually disappear and the rhythm becomes regular, whereas it usually increases the irregularity in auricular

likely to persist It is a sign of left ventricular failure and is of *grave significance* When due to organic heart disease it usually carries a life expectancy of six months When compensation occurs the alternation usually disappears

Rapid tachycardia such as paroxysmal tachycardia is often associated with alternation This alternation disappears when the rate returns to normal This type of alternation is of no significance in itself usually resulting from the fatigue produced by tachycardia Excessive amounts of digitalis may also produce alternation This type of alternation is reversible as cessation of digitalis therapy results in a return to the normal mechanism

The *diagnosis* of pulsus alternans is not difficult (Fig 202) It has been described in detail with the other reliable signs of heart disease With the strong contraction a large volume of blood is ejected and a strong pulsation occurs in the arteries Th
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alternating weak

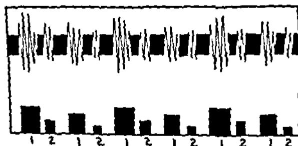


Fig 202 *Pulsus alternans* The heart sounds are alternately intense and feeble the former occurring with the strong contractions and the latter with the feeble ones Consult the text for details

eral arteries When the differences are great enough to be noted by inspection or palpation the blood pressure difference is at least 20 mm of mercury The differences in strength and volume of the alternating pulsation may be observed in the jugular pulsations
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1 Pressure on the carotid sinuses may revert the paroxysmal tachycardia to a normal rhythm but has no effect on auricular fibrillation There fore a return to a normal mechanism following carotid sinus pressure eliminates auricular fibrillation

2 Evidence of auricular contraction obtained by observing *a* waves in the jugular pulsations, auricular or fourth heart sound and a presystolic crescendo murmur rules out auricular fibrillation

3 Runs of absolutely regular rhythm a likely and frequent occurrence even with a variable AV block indicate paroxysmal tachycardia and eliminate auricular fibrillation

The *symptomatology of auricular fibrillation* is essentially the same as that described for auricular flutter and paroxysmal tachycardia and will not be reviewed again

Auricular fibrillation is a sign of heart disease the significance is determined by the underlying cardiac disease Patients may have paroxysmal auricular fibrillation for short periods of time or chronic fibrillation for many years the underlying cardiac state determines the patient s limitations When auricular fibrillation exists for many weeks thrombosis occurs along the walls within the distended paralyzed auricles At times emboli escape producing clinical disturbances determined by the location of the emboli

Treatment —The treatment of auricular fibrillation is similar to that described for auricular flutter and paroxysmal tachycardia except that acetyl beta methyl choline and measures to increase vagal tone are never employed therapeutically When quinidine sulfate fails to arrest the fibrillation digitalis is the drug of choice Quinidine arrests fibrillation more readily if it is of short duration The patient with a rapid ventricular rate or congestive heart failure should receive digitalis according to the method of administration described previously The object is to produce compensation if failure exists to maintain a ventricular rate of 70 to 80 and to reduce the pulse deficit to a minimum Maintenance doses of digitalis should be continued as long as fibrillation exists

Alternation of the Heart

Alternation of the heart consists of alternating strong and weak contractions

The *mechanism* is not well known but it is produced by organic heart disease Because of the disease each alternating beat is associated with a contraction of all of the ventricular musculature the other alternating contraction being associated with a contraction of only a portion of the ventricular muscle When all of the muscle contracts a large volume of blood is ejected with a normal or great force whereas contraction of only a portion of the ventricular muscle is followed by ejection of only a small volume of blood under less pressure

When it is caused by organic heart disease the alternation is more

usually strong The jugular pulse is free from c and v waves at the moment of the AV blocking An a wave is noted since the auricles contract (Fig 205) On auscultation of the heart no first and second sounds are heard when the block occurs as there is no ventricular contraction (Fig 205) In other words there is a 'dropped ventricular beat' The auricular sound may be heard During recording of the arterial blood pressure no Korotkow sound is heard for that cycle in which the auricular impulse is blocked at the AV node and the ventricles fail to contract

If there is a 2 1 block this occurs with every other beat that is one auricular impulse is conducted to the ventricles and initiates a ventricular contraction whereas the alternating one is not (Fig 205) The findings should be obvious for 3 1 4 1 12 11 or any sort of AV block in which an auricular impulse fails to pass the AV node

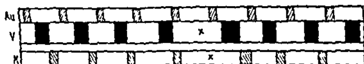


Fig 204 *Wenckebach periods* showing the progressive separation of the atrial and ventricular contractions The arrow indicates failure of the auricular impulse to penetrate the AV node with resultant failure of the ventricles to contract and the arteries to pulsate



Fig 205 *Partial 2 1 AV block* Note absence of ventricular hemodynamic phenomena when each alternate impulse from the atria is blocked at the AV node

3 *Complete heart (AV) block* is not difficult to recognize clinically The findings of diagnostic importance are

a A history of Stokes Adams attacks when the AV block occurs suddenly If true syncope does not occur there is usually an episode of faintness with the onset of the block

b The auricular rate (determined from the a waves in the jugular pulse auricular heart sound *systoles en écho* or pres stol —
is about twice the ventricular ra
or Korotkow sounds) The ve
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Heart Block

Heart block consists of a delay or absence of ventricular response to the auricular impulse

It is produced by impairment of conduction through the AV node, bundle of His or the two main bundle branches Because of this delay or impairment the time relation of auricular and ventricular contractions varies The degree and location of the block also varies The various types of block which may be recognized at the bedside are

1 Incomplete AV block (delayed AV conduction)
2 Partial AV block (2 1 3 1, 4 1 etc)
3 Complete heart (AV) block

The cause of heart block is usually the conduction tissue The common diphtheria myocarditis of any sort anomalies and arteriosclerosis All of these factors produce acute or chronic change in the conduction tissue Infectious diseases fatigue tobacco alcohol and caffeine beverages will precipitate heart block It is also produced by overdigitalization and by overdoses of quinidine

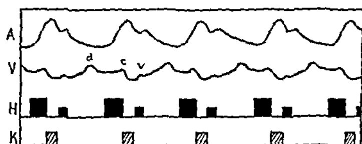


Fig 203 — *Incomplete AV block* with delayed AV nodal conduction Note the wide separation of the *a* wave in the jugular pulse from the *c* and *v* waves

Heart block occurs at any *age* the age incidence varying with the cause Because of the high incidence of arteriosclerosis in individuals after the age of fifty years heart block is more common in this age group It is more common in *men* than in women

The Diagnosis of Heart Block 1 The recognition of *delayed AV conduction* is usually difficult clinically When the heart rate is not rapid and the delay fairly *long* it is *sometimes possible to recognize an a wave* in the jugular pulse which is widely separated from the *c* wave (Fig 203) If there is an auricular heart sound or late diastolic crescendo murmur these may be widely separated from the first heart sound

2 *Partial AV block* is not difficult to recognize In the peripheral arterial pulse a complete pulsation drops out when the impulse from the auricles is blocked at the AV node (Fig 204) The regularly occurring pu felt When the auricular impulse at the AV node is blocked

a

to

visible jerky pulsation of the jugular vein but also a peculiar sensation to the patient.

Heart block of any type is indicative of serious heart disease. Most patients with complete heart block die within a few months, although some patients have lived many years. The *prognosis* should be determined by the underlying cardiac disease and fitness of the heart muscle. The younger patient with good cardiac muscle is more likely to survive many years whereas the old patient with coronary sclerosis is likely to die early and suddenly.

Treatment.—The treatment of heart block consists essentially in:

1. Rest, mental and physical.
2. Proper hygienic measures necessarily important to all people.
3. Proper care of the underlying cardiac disease and its complications, such as congestive heart failure.
4. Management of *persistent block* requires no specific measures. Do not employ drugs that impair AV nodal conduction or depress the cardiac muscle, such as digitalis and quinidine, unless absolutely necessary.
5. The *onset of high grades of heart block*, particularly complete heart block, requires no treatment *per se*. If a physician is present when complete AV block sets in and ventricular asystole exists, the intracardiac injection of 0.5 to 1.0 cc. of epinephrine may save the patient's life. Benzedrine sulfate or ephedrine sulfate should be employed if there are repeated episodes of Stokes-Adams syndrome. Oxygen is of considerable value when the ventricular rate is slow and the circulation is seriously reduced. Patients who are prone to syncope should be warned never to climb to high levels, walk near the edge of boats, or undertake any task where unconsciousness may cost them their lives.

taneously, a 'booming' sound (*bruit de canon*) is heard. At the time of simultaneous auricular and ventricular contraction the auricle cannot eject its blood into the ventricle (its pressure holds the tricuspid valve closed) so that the blood is forced into the superior vena cava and jugular vein. This results in a 'jerking' type of jugular pulsation.

c. The jugular pulsations show *waxing and waning with respiration*. This is strongly suggestive evidence of complete heart block.

The symptoms produced by heart block are the result of

(1) The underlying cardiac disease.
(2) The impaired peripheral circulation.

The factors concerned in (1) have already been discussed. The symptoms produced by the impaired peripheral circulation (2) are of two types:

(*a*) Those due to the *sudden* ventricular slowing.
(*b*) *Those due to the chronically slow ventricular rate.*

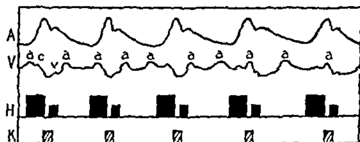


Fig. 206.—Complete AV block. Note the complete dissociation of the atrial and ventricular hemodynamic activity. The atrial hemodynamic events are essentially twice as rapid as the ventricular ones. Study this figure carefully.

When complete AV block occurs there is a period of ventricular standstill. This suddenly results in an inadequate and less than customary supply of blood to the tissues. Since the central nervous system is particularly sensitive to impairment of blood supply, syncope and sometimes convulsions follow. The sudden syncope is known as the *Stokes-Adams syndrome*. The idioventricular rhythm which is established then ensures ventricular contractions at a slow rate. This starts the circulation again. The central nervous system then receives blood, and the patient becomes conscious.

Because of the slow ventricular rate, the total circulation to the tissues is reduced and all tissues function with less efficiency. This is well manifested by skeletal muscle weakness, lethargy, and cerebral anoxia that occur. When the ventricular rate is between 10 and 20, extreme weakness and then unconsciousness supervene. Pallor, stertorous breathing, muscular twitchings, convulsions and death may follow periods of asystole lasting fifteen seconds or more. The convulsions often create diagnostic confusion with *idiopathic epilepsy*.

The patient may be conscious of bradycardia because of the volume and vigor of each ventricular ejection, both explained by Starling's law of the heart. The synchronous contraction of the atria and ventricles with the resultant ejection of the atrial blood into the neck not only produces a

APPENDIX

The Nomenclature and Classification of Heart Disease as advanced by the New York and American Heart Associations The book, "Nomenclature and Criteria for Diagnosis of Diseases of the Heart," is an excellent one which should be studied by every student

Etiological Diagnosis

1 Anemia

6 Hypertension
7 Hyperthyroidism[1]
8 Hypothyroidism
9 Neoplasm
10 Other etiological factors (to be specified)
11 Psychoneurosis
12 Pulmonary disease (to be specified)
13 Reflex action
14 Rheumatic fever[1]
15 Syphilis[1]
16 Thoracic deformity
17 Toxic agent (specify if possible)
18 Trauma
19 Unknown

Anatomical Diagnosis

Diseases of Aorta and Pulmonary Arteries

1 Aneurysm (specify location)
2 Aortitis
3 Arteriosclerosis of aorta
  (a) Without dilatation
  (b) With dilatation

[1] When one of these diagnoses is used it should be stated, if possible whether the etiological factor is still active or is inactive

APPENDIX

The Nomenclature and Classification of Heart Disease as advanced by the New York and American Heart Associations. The book, "Nomenclature and Criteria for Diagnosis of Diseases of the Heart," is an excellent one which should be studied by every student.

Etiological Diagnosis

1 Anemia
2 Arteriosclerosis
3 [illegible]
4
5
6
7 Hyperthyroidism[1]
8 Hypothyroidism
9 Neoplasm
10 Other etiological factors (to be specified)
11 Psychoneurosis
12 Pulmonary disease (to be specified)
13 Reflex action
14 Rheumatic fever[1]
15 Syphilis[1]
16 Thoracic deformity
17 Toxic agent (specify if possible)
18 Trauma
19 Unknown

Anatomical Diagnosis

Diseases of Aorta and Pulmonary Arteries

1 Aneurysm (specify location)
2 Aortitis
3 Arteriosclerosis of aorta
   (a) Without dilatation
   (b) With dilatation
4 Arteriosclerosis of pulmonary arteries
5 Congenital anomaly (specify if possible)
6 [illegible]

[1] When one of these diagnoses is used, it should be stated, if possible, whether the etiological factor is still active or is inactive.

Diseases of Coronary Arteries

13 Arteriosclerosis of coronary arteries
  (a) With narrowing
  (b) With occlusion
14 Arteritis of coronary arteries
15 Congenital anomaly of coronary arteries
16 Embolism of coronary artery
17 Injury of coronary artery (specify character of lesion)
18 Other disease of coronary arteries (specify)
19 Periarteritis nodosa of coronary arteries
20 Stenosis of coronary ostium
21 Thrombosis of coronary artery

Diseases of Myocardium

(Including Conduction System and Heart as a Whole)

22 Aneurysm of heart (specify location)
23 Atrophy of heart
24
25
26
  (a) Dilatation
  (b) Hypertrophy
27 Fatty infiltration of heart
28 Fibrosis of myocardium
29 *Infarct of myocardium*
  (a) Recent
  (b) Healed
30 Injury of heart (specify character of lesion)
31
32
33
34
35
36
37 ossible)

Diseases of Endocardium and Valves

38 (specify lesion if possible)
39
40
41 lenta) (specify organism)
42
43
44
45 Other structural disease (specify location if possible)
46
47
48 location if possible)
49
50
51

(d) Mitral stenosis
(e) Pulmonic insufficiency
(f) Pulmonic stenosis
(g) Tricuspid insufficiency
(h) Tricuspid stenosis

Diseases of Pericardium

5[illegible] Calcification of per[illegible]

[illegible] [illegible]carditis, acute
(a) Fibrinous
(b) Serofibrinous
(c) Suppurative
[illegible]

[illegible] [illegible]erous
60 Pneumopericardium

PHYSIOLOGICAL DIAGNOSIS

Cardiac Mechanism

1 Arrhythmia (undiagnosed)
2 Auricular fibrillation
(a) Paroxysmal
(b) Persistent
3 Auricular flutter
(a) Paroxysmal
(b) Persistent
4 Auriculoventricular block
(a) Prolonged conduction time
(b) Incomplete
(c) Complete
[illegible] Auric[illegible]
[illegible]

(b) Auriculoventricular nodal (junctional)
(c) Ventricular
(d) Unknown origin
9 Premature contractions
(a) Auricular
(b) Auriculoventricular nodal (junctional)
(c) Ventricular
(d) Unknown origin
10 Sinus arrest
11 Sinus arrhythmia
12 Sinus bradycardia
13 Sinus rhythm normal
14 Sinus tachycardia

15 Ventricular escape
16 Ventricular fibrillation
17 Wandering pacemaker
18 Valvular incompetence
  (a) Aortic incompetence
  (b) Mitral incompetence
  (c) Pulmonic incompetence
  (d) Tricuspid incompetence

Clinical Syndromes

19 Adams-Stokes syndrome
20 Anginal syndrome
21 Cardiac insufficiency
22 Carotid sinus syndrome
23 Pulsus alternans
24 Paroxysmal dyspnea
25 Paroxysmal pulmonary edema

FUNCTIONAL CAPACITY

Class I (Formerly I*) Patients with a cardiac disorder* without limitation of physical activity Ordinary physical activity causes no discomfort
Class II (Formerly IIa*) Patients with a cardiac disorder with slight to moderate limitation of physical activity Ordinary physical activity causes discomfort
Class III (Formerly IIb*) Patient with a cardiac disorder with moderate to great limitation of physical activity Less than ordinary physical activity causes discomfort
Class IV (Formerly III*) Patients with a cardiac disorder unable to carry on any physical activity without discomfort

THERAPEUTIC CLASSIFICATION

Class A Patients with a cardiac disorder whose ordinary physical activity needs no restriction
Class B Patients with a cardiac disorder whose ordinary physical activity needs no restriction but who should be advised against unusually severe or competitive efforts
Class C Patients with a cardiac disorder whose ordinary physical activity should be moderately restricted and whose more strenuous habitual efforts should be discontinued
Class D Patients with a cardiac disorder whose ordinary physical activity should be markedly restricted
Class E Patients with a cardiac disorder who should be at complete rest or confined to bed

POTENTIAL HEART DISEASE

Patients without heart disease whom it is advisable to follow because of the presence or history of an etiological factor which might cause heart disease should be diagnosed as Potential Heart Disease In such cases the etiological factor should be stated

* The former classes I IIa IIb and III included only patients with organic heart disease

POSSIBLE HEART DISEASE

Patients with symptoms or signs referable to the heart but in whom a diagnosis of cardiac disease is uncertain should be classified as Possible Heart Disease

RELATIONS OF CARDIAC MEASUREMENTS TO BODY SIZE

Ungerleider and his associates found the cardiac size to be related to body build in such a way as to make certain measurements be of some [illegible] used in [illegible] many [illegible] The following data may prove to be of value

The measurements of (1) *transverse diameter*, (2) *area of the frontal cardiac silhouette* (usually inaccurate because of difficulty in estimating the inferior and superior) (3) *transverse diameter of the silhouette of the frontal aortic arch* are correlated with height and weight of the patient When any of these measurements exceeds 10 per cent of the expected standard value it is considered to be abnormal

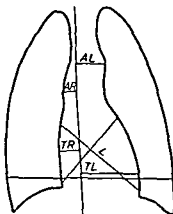


FIG 207 Illustration of the various measurements that may be made when estimating cardiac size (Courtesy of Dr Harry E Ungerleider and the Equitable Life Assurance Society of the United States) Transverse diameter = TR + TL (sum of maximal projections to right and left borders from midline) Long diameter = L (extends from junction of cardiac silhouette and vascular pedicle on right to apex on left) Broad diameter = B (greatest diameter of cardiac shadow perpendicular to long diameter This is sometimes drawn as the sum of two perpendiculars from the long diameter to the right and to the left heart borders For calculation of the cardiac area in the Nomogram the broad diameter should be drawn in a single line as indicated When the heart is transversely placed it may be necessary to extend the lower right heart border in its natural curve to delineate the margin of the broad diameter) Aortic arch diameter = AR + AL (sum of maximal extension to right and to left borders of vascular pedicle of midline)

TABLE 19

THEORETICAL TRANSVERSE DIAMETERS OF HEART SILHOUETTE FOR VARIOUS HEIGHTS AND WEIGHTS

T D of heart mm	Height 5'0"	1"	2"	3"	4"	5"	6"	7"	8"	9"	10"	11"	6'0"	1"	2"	3"	4"	5"	6"
100	83	85	86	87	89	90	92												
101	85	86	88	89	91	92	93	95											
102	87	88	90	91	92	94	95	97											
103	88	90	92	93	94	96	97	99	100										
104	90	92	93	95	96	98	99	101	102										
105	92	93	95	96	98	99	101	103	104	106									
106	94	95	97	98	100	101	103	104	106	108									
107	95	97	99	100	102	103	105	106	108	110	111								
108	97	99	100	102	104	105	107	108	110	112	113								
109	99	101	102	104	106	107	109	110	112	114	115	117							
110	101	102	104	105	108	109	111	113	114	116	118	119	121						
111	103	104	106	108	109	111	113	115	116	118	120	121	123	125					
112	105	106	108	110	111	113	115	117	118	120	122	124	125	127	129				
113	107	108	110	112	113	115	117	119	121	123	124	126	128	129	131	133			
114	108	110	112	114	115	117	119	121	123	125	126	128	130	132	133	135	137		
115	110	112	114	116	117	119	121	123	125	127	129	130	132	134	136	139	140	141	
116	112	114	116	118	120	121	123	125	127	129	131	133	134	136	138	140	142	144	146
117	114	116	118	120	122	124	125	127	129	131	133	135	137	139	141	143	144	146	148
118	116	118	120	122	124	126	128	129	131	133	135	137	139	141	143	145	147	149	151
119	118	120	122	124	125	128	130	132	134	136	138	140	142	143	145	147	149	151	153
120	120	122	124	126	128	130	132	134	136	138	140	142	144	146	148	150	152	154	156
121	122	124	126	128	130	132	134	136	138	140	142	144	146	148	150	152	154	156	159
122	124	126	128	130	132	134	136	138	140	143	145	147	149	151	153	155	157	159	161
123	126	128	130	132	134	135	139	141	143	145	147	149	151	153	155	157	160	162	164
124	128	130	132	134	137	139	141	143	145	147	149	152	154	156	158	160	162	164	166
125	130	132	134	137	139	141	143	145	147	150	152	154	156	158	160	163	165	167	169
126	132	134	137	139	141	143	145	148	150	152	154	156	159	161	163	165	167	170	172
127	134	137	139	141	143	145	148	150	152	154	157	159	161	163	166	168	170	172	175
128	136	139	141	143	145	148	150	152	155	157	159	161	164	166	168	171	173	175	177
129	139	141	143	145	148	150	152	155	157	159	162	164	166	169	171	173	176	178	180

TABLE FOR DETERMINING THE PER CENT DEVIATION FROM AVERAGE

T D of heart mm	Minus 25	20	15	10	5	Av	Plus 5	10	15	20	25
100	75	80	85	90	95	100	105	110	115	120	125
101	76	81	86	91	96	101	106	111	116	121	126
102	77	82	87	92	97	102	107	112	117	122	128
103	77	82	88	93	98	103	108	113	118	124	129
104	78	83	88	94	99	104	109	114	120	125	130
105	79	84	89	95	100	105	110	116	121	126	131
106	80	85	90	95	101	106	111	117	122	127	133
107	80	86	91	96	102	107	112	118	123	128	134
108	81	86	92	97	103	108	113	119	124	130	135
109	82	87	93	98	104	109	114	120	125	131	136
110	83	88	94	99	105	110	116	121	127	132	138
111	83	89	94	100	105	111	117	122	128	133	139
112	84	90	95	101	106	112	118	123	129	134	140
113	85	90	96	102	107	113	119	124	130	136	141
114	86	91	97	103	108	114	120	125	131	137	143
115	86	92	98	104	109	115	121	127	132	138	144
116	87	93	99	104	110	116	122	128	133	139	145
117	88	94	99	105	111	117	123	129	135	140	146
118	89	94	100	106	112	118	124	130	136	142	148
119	89	95	101	107	113	119	125	131	137	143	149
120	90	96	102	108	114	120	126	132	138	144	150
121	91	97	103	109	115	121	127	133	139	145	151
122	92	98	104	110	116	122	128	134	140	146	153
123	92	98	105	111	117	123	129	135	141	148	154
124	93	99	105	112	118	124	130	136	141	149	155
125	94	100	106	113	119	125	131	138	144	150	156
126	95	101	107	113	120	126	132	139	145	151	158
127	95	102	108	114	121	127	133	140	146	152	159
128	96	102	109	115	122	128	134	141	147	154	160
129	97	103	110	116	123	129	135	142	148	155	161

130	141	143	145	148	150	152	155	157	160	162	164	167	169	171	174	176	178	181	183	**130**	94	104	111	117	124	**130**	137	143	150	156	163
131	143	145	148	150	152	155	157	160	162	164	167	169	172	174	176	179	181	183	186	**131**	94	104	111	118	124	**131**	138	144	151	157	164
132	145	148	150	152	155	157	160	162	164	167	169	172	174	177	179	181	184	186	189	**132**	99	103	112	119	125	**132**	139	145	152	158	165
133	147	150	152	155	157	160	162	165	167	169	172	174	177	179	182	184	187	189	192	**133**	100	106	113	120	126	**133**	140	146	153	160	166
134	150	152	155	157	160	162	164	167	169	172	174	177	179	182	184	187	189	192	194	**134**	101	107	114	121	127	**134**	141	147	154	161	168
135	152	154	157	159	162	164	167	169	172	175	177	180	182	185	187	190	192	195	197	**135**	101	108	115	122	128	**135**	142	149	155	162	169
136	154	157	159	162	164	167	169	172	175	177	180	182	185	187	190	193	195	198	200	**136**	102	109	116	122	129	**136**	143	150	156	163	170
137	156	159	162	164	167	169	172	175	177	180	182	185	188	190	193	195	198	201	203	**137**	103	110	116	123	130	**137**	144	151	158	164	171
138	159	161	164	167	170	172	174	177	180	182	185	188	190	193	196	198	201	204	206	**138**	104	110	117	124	131	**138**	145	152	159	166	173
139	162	164	167	169	172	174	177	180	182	185	188	190	193	196	198	201	204	207	209	**139**	104	111	118	125	132	**139**	146	153	160	167	174
140	163	166	169	171	174	177	180	182	185	188	190	193	196	199	201	204	207	209	212	**140**	105	112	119	126	133	**140**	147	154	161	168	175
141	166	168	171	174	177	179	182	185	188	190	193	196	199	201	204	207	210	212	215	**141**	106	113	120	127	134	**141**	148	155	162	169	176
142	168	171	174	176	179	182	185	188	190	193	196	199	202	204	207	210	213	216	218	**142**	107	114	121	128	135	**142**	149	156	163	170	178
143	170	173	176	179	182	184	187	190	193	196	199	202	204	207	210	213	216	219	221	**143**	107	114	122	129	136	**143**	150	157	164	172	179
144	173	176	178	181	184	187	190	193	196	199	201	204	207	210	213	216	219	222	224	**144**	108	115	122	130	137	**144**	151	158	166	173	180
145	175	178	181	184	187	190	193	196	198	201	204	207	210	213	216	219	222	225	228	**145**	109	116	123	131	138	**145**	152	160	167	174	181
146	178	180	183	186	189	192	195	198	201	204	207	210	213	216	219	222	225	228	231	**146**	110	117	124	131	139	**146**	153	161	168	175	183
147	180	183	186	189	192	195	198	201	204	207	210	213	216	219	222	225	228	231	234	**147**	110	118	125	132	140	**147**	154	162	169	176	184
148	182	185	188	192	195	198	201	204	207	210	213	216	219	222	225	228	231	234	237	**148**	111	118	126	133	141	**148**	155	163	170	178	185
149	185	188	191	194	197	200	203	206	210	213	216	219	222	225	228	231	234	237	240	**149**	112	119	127	134	142	**149**	156	164	171	179	186
150	187	191	194	197	200	203	206	209	212	215	219	222	225	228	231	234	237	240	243	**150**	113	120	128	135	143	**150**	158	165	173	180	188
151	190	193	196	199	203	206	209	212	215	218	222	225	228	231	234	237	241	244	247	**151**	113	121	128	136	143	**151**	159	166	174	181	189
152	192	196	199	202	205	208	212	215	218	221	224	228	231	234	237	241	244	247	250	**152**	114	122	129	137	144	**152**	160	167	175	182	190
153	195	198	201	205	208	211	214	218	221	224	227	231	234	237	240	244	247	250	253	**153**	115	122	130	138	145	**153**	161	168	176	184	191
154	198	201	204	207	211	214	217	221	224	227	230	234	237	240	244	247	250	253	257	**154**	116	123	131	139	146	**154**	162	169	177	185	193
155	200	203	207	210	213	217	220	224	227	230	233	237	240	243	247	250	253	257	260	**155**	116	124	132	140	147	**155**	163	171	178	186	194
156		206	210	213	216	220	223	227	230	233	236	240	243	247	250	254	257	260	264	**156**	117	125	133	140	148	**156**	164	172	179	187	195
157				216	219	222	225	229	232	236	239	243	246	250	253	257	260	263	267	**157**	118	125	133	141	149	**157**	165	173	181	188	196
158						225	229	232	236	239	243	246	249	253	256	260	263	267	270	**158**	119	126	134	142	150	**158**	166	174	182	190	198
159								235	239	242	246	249	253	256	260	263	267	270	274	**159**	119	127	135	143	151	**159**	167	175	183	191	199
160										245	249	252	256	259	263	266	270	274	277	**160**	120	128	136	144	152	**160**	168	176	184	192	200
161												255	259	263	266	270	273	277	281	**161**	121	129	137	145	153	**161**	169	177	185	193	201
162												259	262	266	270	273	277	280	284	**162**	122	130	138	146	154	**162**	170	178	186	194	203
163														269	273	277	280	284	288	**163**	122	130	139	147	155	**163**	171	179	187	195	204
164														273	276	280	284	287	291	**164**	123	131	139	148	156	**164**	172	180	188	197	205

PREDICTED AREA FROM WEIGHT AND HEIGHT AND ACTUAL AREA FROM LONG AND BROAD DIAMETERS

(A = 5 L × B) FOR ORTHODIAGRAM AND TELEOROENTGENOGRAM

BROAD DIAMETER CM ORTH TELE

FRONTAL AREA SQ CM ORTH TELE

HEIGHT IN CM

WEIGHT LBS KG

LONG DIAMETER CM ORTH TELE

TRANSVERSE DIAMETER OF HEART AND AORTIC SILHOUETTE PREDICTED FROM WEIGHT AND HEIGHT

FOR TELEOROENTGENOGRAM

TRANSVERSE DIAMETER (MM) HEART AORTA*

WEIGHT LBS KG

HEIGHT IN CM

*FOR AORTIC DIAMETER ADD MM FOR EACH 5 YRS OVER AGE [illegible] AND SUBTRACT MM FOR EACH 3 YRS UNDER [illegible]

FIG 208 —Nomogram for predicting the cardiac area and body weight and height ... The values for actual (or predicted) area are read at the point at which

TABLE 20 — MISCELLANEOUS NORMAL CARDIAC MEASUREMENTS

B Weight of Average Normal Heart
1 At birth 20 grams
2 6 months 24 gram
3 1 year 30 grams
4 2 years 45 grams
5 4 years 70 grams
6 9 years 100 grams
7 15 years 200 grams
8 Adult Male 310 grams
Adult Female 255 grams

C Relationship of Heart Weight to Body Weight
1 The heart forms 0 4–0 45 per cent of total body weight in male of normal build
2 The heart forms 0 ,33 per cent of total body weight in female of normal build

D Heart Volume
1 At birth 22 cc
2 15 years 155 cc
3 20 years 250 cc
4 50 years 280 cc

E Thickness of Wall of Average
I RA 2 mm
II LA 3 mm
III RV 2–4 mm
IV LV 10–14 mm

F Capacity of Average
I RA 57 cc
II LA 125 cc
III RV 85 cc
IV LV 85 cc

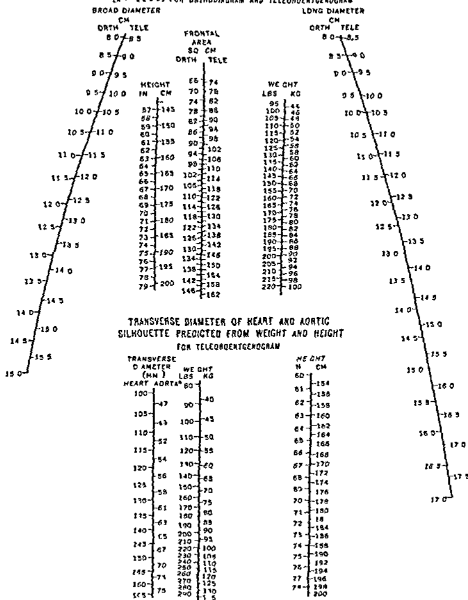
G Circumference of Valves of Normal Heart
I Mitral 10 cm
II Aortic 7 cm
III Tricuspid 12 cm
IV Pulmonary 8 cm

DIETS EMPLOYED IN CARDIAC DISEASE

It [illegible]
of ca
are t [illegible] New Orleans

It [illegible] to remember that there is relatively little sodium in animal and plant tissue Therefore under ordinary circumstances food cooked without any added salt is low in sodium Salty foods such as smoked [illegible] salted meats bacon [illegible]

Substitutes for
not palatable



... and height
at which
id height)
oentgeno-
iameter of

Rice Diet

Name

Date

Approx 2400 Calories
30 gm protein
10 to 100 mg sodium
750 cc Fluids

Food	Amount
Breakfast.	
Steamed Rice (cooked without salt)	1¾ cup steamed (3 oz raw)
Fruit Juice	½ cup (4 oz)
3 Choices of Fruit List R	See List
Sugar (White)	4 level teaspoons
Honey	4 level teaspoons
Mid Morning	
Fruit Juice	½ cup (4 oz)
Lunch	
Steamed Rice (cooked without salt)	1¾ cup steamed (3 oz raw)
3 Choices of Fruit List R	See List
Fruit Juice	½ cup (4 oz)
Sugar (White)	4 level teaspoons
Honey	4 level teaspoons
Mid Afternoon	
Fruit Juice	½ cup (4 oz)
Dinner	
Steamed Rice (cooked without salt)	1¾ cup steamed (3 oz raw)
Fruit Juice	½ cup (4 oz)
3 Choices of Fruit List R	See List
Sugar (White)	4 level teaspoons
Honey	4 level teaspoons
Bedtime	
Fruit Juice	½ cup (4 oz)

Rice Diet

General Instructions

1 ...

2 ... brown rice or wild rice may be used It may be boiled or steamed in plain water or fruit juice without salt, milk or fat The palatability of the rice depends to some extent upon the way in ... ordinary ... about two ... hot water ... This meth... wet rice ...

... is omitted

Karrell Diet

The Karrell Diet is used in acute nephritis or acute cardiac failure Caloric and nutritional requirements are ignored temporarily The diet consists entirely of milk or fruit juice if patient cannot tolerate milk

8 00 A M	200 cc milk or fruit juice
12 00 NOON	200 cc milk or fruit juice
5 00 P M	200 cc milk or fruit juice
8 00 P M	200 cc milk or fruit juice
Total	800 cc milk or fruit juice

40 gm CHO
26 gm Prot
32 gm Fat
550 Calories
1 6 gm NaCl

This diet should not be given any longer than five to seven days If the patient s condition is still severe, additions should be made to the diet, and a modification of the Karrell Diet should be used

Karrell Diet With Modifications

(Carbohydrate 198, Protein 45, Fat 32, Calories 1260, Salt 3 6 gm)

Cooked or canned fruit
Soft cooked eggs
Custards
Jello

Sample Menu

8 00 A M
200 cc milk
Cereal with sugar

12 00 NOON
200 cc milk
Toast and jelly

4 00 P M
200 cc milk
Toast
Cereal with sugar

8 00 P M
200 cc milk
Stewed fruit with sugar

Salt Restricted Diets

In cases of cardiac and renal disorders when edema is present or threatened salt must be restricted or eliminated entirely from the diet Fluid limitations may also be necessary, however, recent studies have shown that forcing fluids in the presence of edema may be a beneficial procedure

Cardiac Diets

1
2
3

Nephritic Diets

1 Karrell—acute nephritis
2 Modified Karrel—acute nephritis

Charity Hospital of Louisiana Clinics

Restricted Sodium Diet I
Protein—70 gms Sodium—.3 .5 gms

Foods Daily	*You May Eat*	*Do Not Eat*
Meats (1 or 2 servings)	Fresh beef, veal, mutton lamb liver, tongue kidney tripe, wild game chicken, turkey fresh water fish	Smoked cured canned or pickled meat or fish, sausage ham bacon salt pork dried beef, shell fish, salt water fish
Cheese	Creole cream cheese or cottage cheese may be used instead of meat Once a day	American, Swiss or Philadelphia cream cheese
Egg (one)	1 or more a day	Fried eggs
Fruit (citrus fruit)	Any juice or fruit	Dried figs peaches apricots, and raisins
Vegetable (1 green and 1 raw)	Fresh or frozen vegetables	Any prepared with added salt frozen peas or lima beans sauerkraut pickles, canned vegetables celery beets
Potato (as desired)	Potato macaroni rice, grits spaghetti (all prepared without salt)	Potato chips fried potatoes
Cereal and bread (3 servings)	Salt free cooked cereals puffed rice puffed wheat shredded wheat salt free bread	All other dry cereals salted crackers cornbread biscuits anything with baking powder or baking soda all bakery goods
Fat (as desired)	Washed butter or margarine oil all unsalted fats	Bacon fat salted dressing salt pork fried foods
Dessert (as desired)	Gelatin desserts fruits pud dings custard	Desserts made with salt baking soda or baking powder
Sweets (as desired)	Sugar honey syrup hard candy jelly jam	Jams and jellies prepared with sodium benzoate candy bars
Soup (as desired)	Homemade only	Canned soup bouillon
Beverage (1 cup milk)	Coffee tea 1 cup milk home-made buttermilk lemonade	Dairy buttermilk
Miscellaneous	Spices vinegar unsalted nuts herbs unsalted salad dressings	Salt olives mustard catsup chili meat sauce peanut butter, salted nuts

Sample Menu

Breakfast	*Dinner*	*Supper*
Orange juice	Meat Stew	Cream Cheese
Oatmeal	Rice or Potato	Baked Potato
Egg	Greens	Sliced Tomatoes
Salt Free Bread and Butter	Stewed Prunes	Fruit
Coffee	S F Bread and Butter	S F Bread and Butter
	Milk	

3 Combinations of rice fruits and sugar may be varied according to taste Some patients who object to the sweet taste find the use of lemon juice helpful
4 A standard measuring cup (8 oz) is used
5 Usually water is omitted and fluid is limited from 700 to 1,000 cc of fruit juice per day
6 The caloric intake varies according to whether weight gain or weight loss is desired in the individual patient

Rice Pudding

¾ cup cooked washed rice
One portion of fruit from Fruit List R

Combine the rice and fruit Moisten with part of allowed fruit juice Season to taste with sugar or honey Bake in ramekins

Frozen Fruit Cup

1 Tablespoon cooked washed rice
One portion of fruit from Fruit List R
¾ cup of allowed fruit juice

Combine rice fruit and juice Season to taste with sugar Freeze

Rice Beverage (1)

6 Tablespoons cooked washed rice
One portion of fruit from Fruit List R
½ cup of allowed fruit juice

Combine rice fruit and juice Season to taste with sugar or honey Blend until smooth

Rice Beverage (2)

6 Tablespoons cooked washed rice
¾ cup of allowed fruit juice

Combine rice and fruit juice Season to taste with sugar or honey Blend until smooth

Fruit List R

One average portion of the following fruits may be used

Apple fresh or stewed sweetened
Apricots fresh or stewed sweetened
Banana (once a day)
Blackberries fresh or stewed sweetened
Blueberries fresh or stewed sweetened
Cantaloupe (one-eighth per day)
Cherries fresh or stewed sweetened
Cranberry jelly (homemade)
Cranberry sauce
Figs fresh or stewed sweetened
Grapefruit fresh
Grapes
Honey Dew Melon (one-eighth per day)
Lemon
Lime
Orange fresh
Peach fresh or stewed sweetened
Pear fresh or stewed sweetened
Pineapple fresh or stewed sweetened
Plum fresh or stewed sweetened
Raspberries fresh or stewed sweetened
Rhubarb stewed sweetened
Strawberries
Tangerine
Watermelon

Check with the typical day as to amount of fruit juice allowed in your diet in a day You may choose from the following fruit juices

Apple juice
Cranberry juice (homemade)
Grapefruit juice
Grape juice
Lemon juice
Orange juice
Lime juice
Pineapple juice

Charity Hospital of Louisiana Clinics

Restricted Sodium Diet 1
Protein—70 gms Sodium—.3 .5 gms

Foods Daily	*You May Eat*	*Do Not Eat*
Meats (1 or 2 servings)	Fresh beef, veal, mutton, lamb, liver, tongue kidney, tripe, wild game chicken, turkey, fresh water fish	Smoked, cured canned or pickled meat or fish, sausage ham, bacon, salt pork, dried beef, shell fish, salt water fish
Cheese	Creole cream cheese or cottage cheese may be used instead of meat ONCE a day	American, Swiss or Philadelphia cream cheese
Egg (one)	1 or more a day	Fried eggs
Fruit (citrus fruit)	Any juice or fruit	Dried figs, peaches, apricots, and raisins
Vegetable (1 green and 1 raw)	Fresh or frozen vegetables	Any prepared with added salt, frozen peas or lima beans sauerkraut, pickles, canned vegetables, celery, beets
Potato (as desired)	Potato, macaroni, rice, grits, spaghetti (all prepared without salt)	Potato chips, fried potatoes
Cereal and bread (3 servings)	Salt free cooked cereals, puffed rice, puffed wheat, shredded wheat, salt free bread	All other dry cereals, salted crackers, cornbread, biscuits, anything with baking powder or baking soda, all bakery goods
Fat (as desired)	Washed butter or margarine, oil, all unsalted fats	Bacon fat salted dressing, salt pork, fried foods
Dessert (as desired)	Gelatin desserts, fruits, puddings, custard	Desserts made with salt, baking soda or baking powder
Sweets (as desired)	Sugar, honey, syrup hard candy, *jelly, jam*	Jams and jellies prepared with sodium benzoate candy bars
Soup (as desired)	Homemade, only	Canned soup, bouillon
Beverage (1 cup milk)	Coffee tea 1 cup milk homemade buttermilk, lemonade	Dairy buttermilk
Miscellaneous	Spices, vinegar unsalted nuts herbs unsalted salad dressings	Salt olives mustard, catsup, chili meat sauce peanut butter, salted nuts

Sample Menu

Breakfast	*Dinner*	*Supper*
Orange juice	Meat Stew	Cream Cheese
Oatmeal	Rice or Potato	Baked Potato
Egg	Greens	Sliced Tomatoes
Salt Free Bread and Butter	Stewed Prunes	*Fruit*
Coffee	S F Bread and Butter	S F Bread and Butter
	Milk	

3 Combinations of rice fruits and sugar may be varied according to taste

4

5

6 ing to whether weight gain or weight loss is

Rice Pudding

¾ cup cooked washed rice
One portion of fruit from Fruit List R

Combine the rice and fruit

Frozen Fruit Cup

1 Tablespoon cooked washed rice
One portion of fruit from Fruit List R
¾ cup of allowed fruit juice

Combine rice fruit and juice Season to taste with sugar Freeze

Rice Beverage (1)

6 Tablespoons cooked washed rice
One portion of fruit from Fruit List R
½ cup of allowed fruit juice

Rice Beverage (2)

6 Tablespoons cooked washed rice
¾ cup of allowed fruit juice

Fruit List R

One average portion of the following fruits may be used

Apple fresh or stewed sweetened
Apricots fresh or stewed sweetened
Grapefruit fresh
Grapes
Honey Dew Melon (one-eighth per day)

Lemon
Lime
Orange fresh
Peach fresh or stewed sweetened
Pear fresh or stewed sweetened
Pineapple fresh or stewed sweetened
Plum fresh or stewed sweetened
Raspberries fresh or stewed sweetened
Rhubarb stewed sweetened
Strawberries
Tangerine
Watermelon

Check with the typical day as to amount of fruit juice allowed in your diet in a day You may choose from the following fruit juices

Apple juice
Cranberry juice *(homemade)*
Grapefruit juice
Grape juice

Lemon juice
Orange juice
Lime juice
Pineapple juice

Low Salt Normal Protein Diet

(Protein 75 gm., NaCl 1 7 gm)

The low salt diet with adequate protein allowance is a diet frequently used in cases of cardiac diseases or chronic nephritis in the absence of albuminuria or edema

Foods Allowed

Bread Without salt, if possible

Cereals As desired, but cooked without salt Oatmeal or other whole grain cereals are preferred No prepared cereals except puffed rice and puffed wheat

Eggs One

Meats One average serving or two small servings of fresh lean meat such as beef lamb chicken or veal Cream cheese or eggs may be used sometimes in place of one small serving of meat

Potatoes One serving Potatoes should be used in preference to macaroni spaghetti white rice or grits Brown rice is just as good as potatoes

Vegetables Two at least besides potatoes and dried beans *Do not cook with salt or salt meat* Try to have one vegetable raw every day

Fruits Two at least Have one raw fruit every day, an orange or grapefruit if possible

Milk One pint (for a grown person)

Butter Without salt, if possible

Sweets fats and other foods may be added to the list above, but they must not have salt in them or taste salty

Sample Menu

Breakfast	*Dinner*	*Supper*
Orange	Veal chop	Cream cheese
Oatmeal	Rice	Baked potato
Eggs	Greens	Sliced tomato
Whole wheat toast	Snap beans	Tapioca pudding
Butter	Stewed prunes	Bread and butter
Coffee if desired	Bread and butter	Milk
	Milk	

Foods to Avoid

Salt in any form

Bicarbonate or baking soda

Fried or greasy foods

Cured salt or smoked meats or fish or bacon

Gas forming fruits and vegetables

Rich pies and pastries

Condiments

Bottled and alcoholic beverages

Soups sauces gravies

Low Salt Soft Diet

(Carbohydrate 185, Protein 57, Fat 59, Calories 1499, NaCl 1 7 gm)

The low salt-soft diet is an inadequate diet and should be used for a limited time only It is low in protein, iron and vitamin B complex

[illegible] to [illegible] of [illegible] up [illegible]

Food Allowed

[illegible] beef white meat of chicken and [illegible]

[illegible] Use potatoes in preference to grits rice, macaroni, etc May be boiled buttered, creamed mashed

Vegetables 2 servings daily—cooked and put through a sieve
Use carrots beets, green beans spinach asparagus tomatoes green peas squash

Fruits 2 servings daily One must be orange or orange juice or grapefruit juice All must be cooked except oranges and bananas Dried fruits must be put through a sieve

Milk *Pasteurized* 1 pint daily for adults 1 quart for children

Desserts Simple puddings jello custard all prepared without salt

Condiments None May use lemon juice for flavor

Beverages 1 cup tea or coffee daily No bottled beverages

Sample Menu

Breakfast	*Dinner*	*Supper*
Orange	Cream cheese	Poached egg
Oatmeal	Mashed potatoes	Grits
1 soft cooked egg	Spinach puree	Beet puree
Toast and butter	Jello	Bread and butter
1 cup weak coffee	Bread and butter	Canned pears
90 cc milk	1 glass milk	1 glass milk

Foods to Avoid

Salt in any form
Bicarbonate or baking soda
Fried or greasy foods
Cured salt or smoked meats
Gas forming vegetables and fruit
Bran
Rich pies and pastries
Condiments
Bottled and alcoholic beverages
Soups, sauces and gravies

Low Salt High Protein Diet

(Protein 100 gm, NaCl 2 7 gm)

A low salt diet high in protein may be indicated

Foods Allowed

6 slices

No prepared cereals, except puffed

Eggs 2-3 daily Not fried

Meat Fish Poultry 2 large servings daily No ham bacon, or salty fish

Cheese Cream cheese only, may be substituted for 1 serving of meat

Potatoes and Substitutes 1 2 times daily Use potatoes in preference to grits macaroni rice etc May be mashed boiled, creamed not fried

Vegetables

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prepared *without*

Sample Menu

Breakfast	*Dinner*	*Supper*
Orange	Scalloped chicken and noodles	Large serving beef
Oatmeal with sugar and milk	Green beans	Mashed potatoes
2 scrambled eggs	Grapefruit salad	Carrots
1 cup weak coffee	Custard	Head lettuce salad with lemon juice
Toast and butter	Milk	Apple
1 glass milk	Bread and butter	Bread and butter
		1 glass milk

Foods to Avoid

Salt in any form
Bicarbonate or baking soda
Fried or greasy food

Bottled and alcoholic beverages
Soups sauces gravies

Acid Ash Diet

Excess Acid Ash over Alkaline Ash 35 cc

(Carbohydrate 195 Protein 100 Fat 110 Calories 2150)

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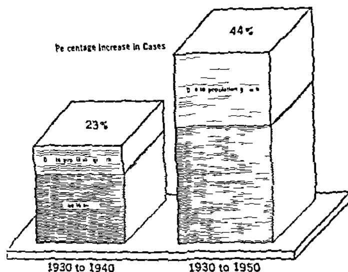
Restricted Sodium Diet 2

Protein 80 gms Sodium 1.5-2 gms

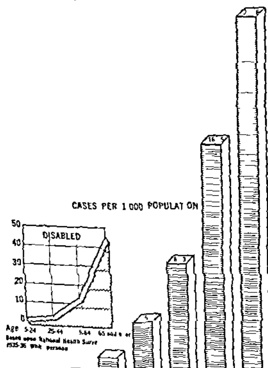
Foods Daily	*You May Eat*	*Do Not Eat*
Meats (1 or 2 servings)	Fresh beef, veal, mutton, lamb, liver, tongue, kidney, tripe, wild game, chicken, turkey, fish and other fresh seafoods	Smoked, cured, canned or pickled meat or fish, sausage, ham, bacon, salt pork, dried beef
Cheese	Creole cream cheese or cottage cheese may be used instead of meat Once a day	American, Swiss or Philadelphia cream cheese
Egg (one)	1 or more a day	Fried eggs
Fruit (citrus fruit)	Any juice or fruit	Dried figs, peaches, apricots and raisins
Vegetable (1 green and 1 raw)	Fresh or frozen vegetables, canned not more than 2 or 3 times a week	Any prepared with added salt, frozen peas or lima beans, sauerkraut, pickles
Potato (as desired)	Potato, macaroni, rice, grits, spaghetti (all prepared without salt)	Potato chips, fried potatoes
Cereal and Bread (3 servings)	Salt free cooked cereals, puffed rice, puffed wheat, shredded wheat, whole grain or enriched bread	All other dry cereals, salted crackers, cornbread, biscuits, anything with baking soda or baking powder
Fat (as desired)	Washed butter or margarine, oil, all unsalted fats	Bacon fat, salted dressing, salt pork, fried foods
Dessert (as desired)	Gelatin desserts, fruits, puddings, custard	Desserts made with salt, baking soda or baking powder
Sweets (as desired)	Sugar, honey, syrup, hard candy, jelly, jam	Jams and jellies prepared with sodium benzoate, candy bars
Soup (as desired)	Homemade only	Canned soup, bouillon
Beverage ($1\frac{1}{2}$ pt. milk)	Coffee, tea, $1\frac{1}{2}$ pt. milk, homemade buttermilk, lemonade	Dairy buttermilk
Miscellaneous	Spices, vinegar, unsalted nuts, herbs, unsalted salad dressings	Salt, olives, mustard, catsup, chili, meat sauce, peanut butter, salted nuts

Sample Menu

Breakfast	*Dinner*	*Supper*
Orange juice	Meat Stew	Cream Cheese
Oatmeal	Rice or Potato	Baked Potato
Egg	Greens	Sliced Tomatoes
Bread and Butter	Stewed Prunes	Fruit
Coffee	Bread and Butter	Bread and Butter
Milk	Milk	Milk



9 *—About 4 000 000 people in the United States are estimated to have heart The inc dence has been increasing because of (1) The aging of the population n rease in the total population



To produce an acid pH of the urine the diet should yield at least 35 cc excess acid ash When the desired pH is acquired, an excess of 17 cc acid ash is sufficient to maintain a low pH

Fluids should be forced, and the salt content of the diet should be low as possible No salt should be used either during cooking or at the table

Foods Allowed

Bread Use freely—any kind except quick breads as biscuits, fruit breads

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Cheese Use only unsalted cottage cheese or cream cheese

Potato or Potato Substitutes No potatoes allowed Rice, noodles, macaroni, spaghetti may be used freely

Butter or Margarine As desired—unsalted

Desserts Allowed fruits tapioca, cornstarch, bread, rice puddings, ice cream sugar cookies, plain cakes Avoid all rich pastries and any dessert made with baking soda

Soups Avoid

Beverages Tea, coffee, postum No fruit juices, no bottled or carbonated beverages

Condiments

Accessory Foo sugar candy (no chocolate) may be used

SAMPLE MENU

Breakfast	*Dinner*	*Supper*
Prunes	Roast lamb	Beef patty
Oatmeal with cream and sugar	Buttered rice	Escalloped noodles
Scrambled egg	Small serving lettuce with salt free mayonnaise	Corn
Whole wheat toast and butter	Plain cake	Plums
Coffee or tea with cream and sugar	Whole wheat bread and butter	Whole wheat bread and butter
	Milk—1 glass	Milk—1 glass

Foods to Avoid

1 luncheon meats pickled or cured meats

allowed in above list

2

3 Potatoes

rich pies and pastries All fruit desserts

4

beverages

5

6 All highly basic

Almonds	Olives
Beet greens	Parsnips
Dandelion greens	Raisins
Figs	Spinach
Molasses	Dried fruits and vegetables

Charity Hospital of Louisiana Clinics

Obesity Diet (Low Sodium—931.6 mgms. daily)

1200 Calories C160—P60—F40

Breakfast

Fruit	1 orange, ½ grapefruit or unsweetened orange or grapefruit juice
Bread	1 slice
Egg	1 boiled or poached—*not fried*
Milk, skimmed	1 glass
Margarine or butter	1 level teaspoon
Tea or Coffee	1 cup if desired. Use *no* sugar or cream

Dinner and Supper

Meat, Fish, Chicken	1 large piece of lean fresh meat. Use lean beef, veal, lamb, liver, tongue, kidney, tripe, rabbit, squirrel, wild game. Two eggs, ¼ cup cream cheese may be used in place of the meat
Vegetable Group I	As desired (see list below)
Vegetable Group II	½ cup (see list below)
Fruit	1 serving (see list below)
Milk, skimmed	1 glass
Bread	1 slice or ½ cup of a cooked starchy food
Margarine or butter	1 level teaspoon

Vegetable List

Group I		*Group II*
Asparagus	Merlitons	Carrots
Broccoli	Mushrooms	Beets
Cabbage	*Okra*	Onions
Cauliflower	Radishes	Pumpkin
Celery	Snap Beans	Squash, Yellow
Cucumbers	*Spinach*	Turnips
Eggplant	Squash, White	Peas
Green Peppers	Tomatoes	
Greens (all)		
Lettuce		

Fruit List

1 Apple	1 Pear
3 Apricots	4 *Plums*
½ Banana	2 Prunes
¼ Cantaloupe	1 cup Berries
3 Fresh Figs	1 Tangerine, Satsuma or Manderine
½ Grapefruit	½ Slice Watermelon
1 Orange	
1 Peach	

Rules

1. Eat three meals a day, using only the foods *listed* on the diet. (Eat No Sweets)
2. The milk you use may be fresh milk, buttermilk, canned evaporated milk mixed with an equal *part of* water or powdered skimmed *milk* (1 cup powdered milk to 1 quart of water)

3 Do Not Fry Anything Boil, roast or broil meat Make no roux gravies
4 Do Not Add Sugar or Flour to Anything Saccharin, Crystallose, or Sucryl may be used for sweetening
5 Vegetables—cook in plain unsalted water Never use cooking soda Use No Grease or Fat Salt Meat to season the vegetables Cook vegetables no longer than 10–25 minutes in a small amount of water Use the vegetable water too
6 Drink eight glasses of fluid each day Do not drink bottled drinks
7 These things you may have whenever you like—coffee, tea, lemonade, or broth without fat
8 You may use pepper, all spices, garlic, and vinegar for seasonings Use No Salt
9 Starchy foods which may be used instead of bread are Corn rice potatoes grits, spaghetti, dried beans or peas cooked cereals and puffed rice, puffed wheat and shredded wheat

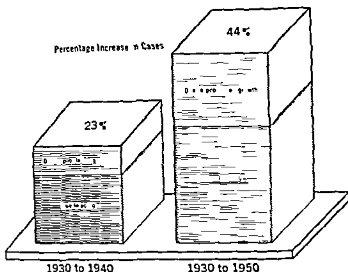
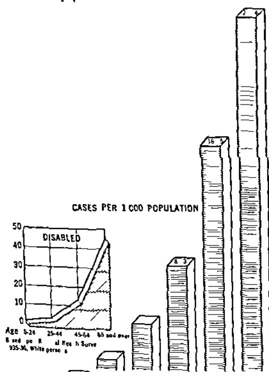


FIG 209.*—About 4 000 000 people in the United States are estimated to have heart disease The incidence has been increasing because of (1) The aging of the population, 2) the increase in the total population.



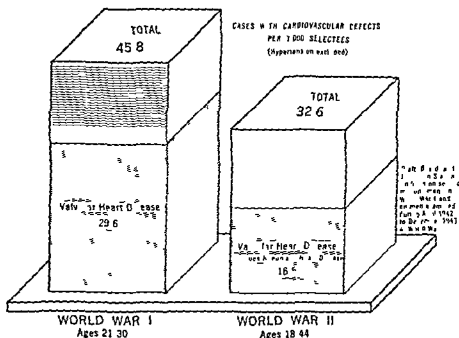


Fig 211 —At the younger ages a sharp reduction in the prevalence rate particularly for the rheumatic heart disease is indicated by Selective Service statistics for World Wars I and II

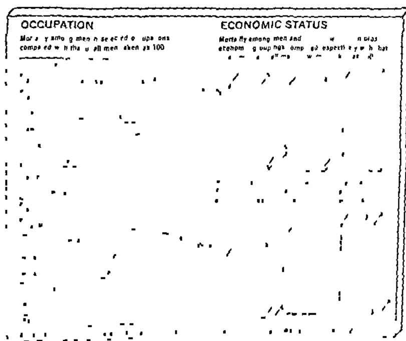


Fig 212 —Distinct occupational differences are found in heart disease among men reflecting physical and mental stresses of occupations This effect is often obscured ... the shifting of ... the influence ... among men

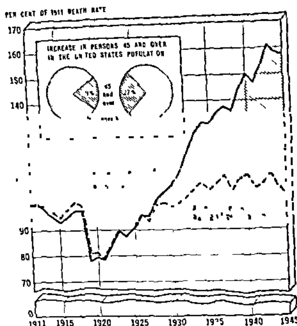


Fig 213 Much of the increase in deaths ascribed to heart disease is accounted for by the advanced aging of the population

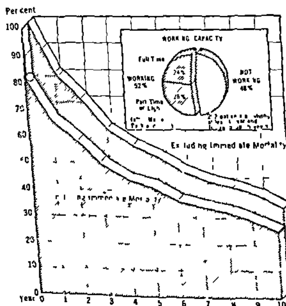


Fig 214 —The greater majority of patients survive the initial attack and most live for many years A large proportion of them are even able to resume normal or near normal activities.

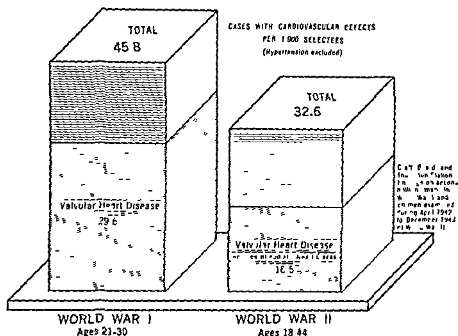
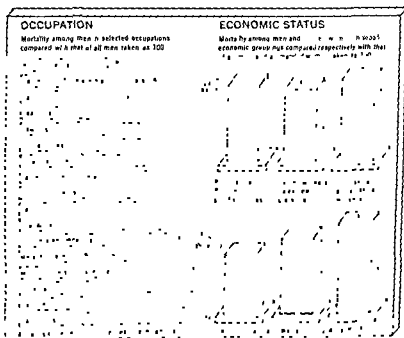


Fig 211 —At the younger ages a sharp reduction in the prevalence rate, particularly for the rheumatic heart disease, is indicated by Selective Service statistics for World Wars I and II



heart disease among men
us effect is often obscured
is well as by the shifting of
patients with heart disease to such occupations ecause of these factors, the influence of economic status tends to be seen more clearly among married women than among men

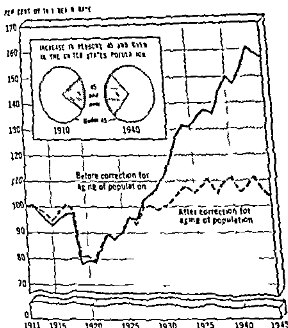


FIG 213 Much of the increase in deaths ascribed to heart disease is accounted for by the advanced aging of the population

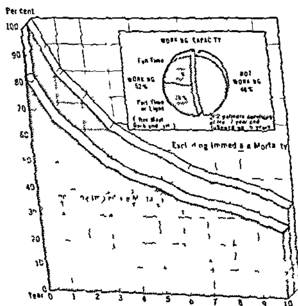


FIG 214 The greater majority of patients survive the initial attack and most live for many years. A large *proportion of* them are even able to resume normal or near normal activities.

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